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TYPHOID FEVER IN SOUTH AFRICA

TREATMENT OF 215 CASES WITHOUT CHLOROMYCETIN AND 139 WITH CHLOROMYCETIN (CHLORAMPHENICOL)

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As a result of a decision by the Union Health Department to pay in part for Chloromycetin used in the treatment of typhoid fever, the hospitals concerned were requested to submit bed letters and other relevant data. Unfortunately a large number of records supplied were inadequate and had to be discarded, but reasonable records of 139 cases treated with Chloromycetin in 1950, in various parts of the Union were obtained in this manner. No doubt the standard of clinical recording in certain hospitals will improve as more facilities are provided. The control group of 215 was obtained from hospitals in the Northern Transvaal, for the period 1948-49, before the introduction of Chloromycetin. The records were taken in order of occurrence, and no selection was exercised, except that a reasonable minimum of completeness of description was necessary for them to be of any value at all.

In by far the majority of cases the diagnosis of typhoid fever was based on clinical impressions and Widal tests, and in only a minority of cases on bacteriological studies of blood, urine and faeces.

From a study of our records it is clear that far more expeditious and certain diagnoses of typhoid fever would be made if laboratory services were more readily available in certain parts of the country. The Widal test alone cannot be relied on for satisfactory diagnostic results and the tendency for a higher percentage of deaths than normal to occur amongst Widal negative cases should be remembered (Armas Cruz *et al.*[1])—in our control series there were five deaths out of the nine who were recorded as Widal negative. It has also been contended by certain of those who have supplied us with records that the early administration of Chloromycetin prevents the Widal turning positive during the illness, but this is impossible to prove or disprove when the diagnosis is based on clinical grounds only. Yet another factor complicating the interpretation of the Widal test in acute typhoid fever is the increasing number of persons who have been immunized against the disease (Lewin [2]), though this is as yet of little significance amongst our rural native population.

In regard to the diagnosis of typhoid fever, Bock [3] has recently shown that sternal puncture for marrow culture gave positive results in 90% of cases, compared with 60% of blood cultures. In this country sternal marrow punctures have also been found useful (Hirsowitz and Goldberg [4]).

THE PRESENT MATERIAL

A. RACE AND SEX

TABLE I

	Treated with Chloromycetin			*Not treated with Chloromycetin*		
	Male	*Female*	*Total*	*Male*	*Female*	*Total*
Europeans	10	3	13	18	9	27
Natives ..	47	70	117	110	75	185
Indians ..	1	2	3	2	1	3
Coloured ..	3	3	6	—	—	—
All races ..	61	78	139	130	85	215

The bulk of our cases (85%) were thus Bantu and 46% were females, which agrees very closely with the 45% reported by Armas Cruz *et al.*[1] from Chile.

B. AGE

As Natives form the majority of cases, we have analysed their age composition. In the group not treated with Chloromycetin the mean age came to 21 years (S.D. ± 19.3, S.E. ± 1.4) with 58.3% under the age of 20. In the group treated with Chloromycetin, the mean age came

to 19 years (S.D. ± 12, S.E. ± 1.22) with 64.3% under the age of 20. It has been well established that typhoid is predominantly a disease of young people.

C. Dosage of Chloromycetin

In the present series, the mean dose of Chloromycetin was 14.60 gm. (S.D. ± 8.30, S.E. ± 0.70, coefficient of variation 56.8%). As shown above, our group included a large number of children who needed a smaller dose than an adult would, but if only adults are included (i.e. those over 16 years of age) the average dose per case was still only 16 gm., which is half the dose for adults (32 gm.) recommended by Smadel *et al.*[5] Pfeiffer[6] recommends a doze of 38.25 gm. for adult cases according to the following schedule.

0.50 gm. 2-hourly for about four days ...	24.00 gm.
0.50 gm. 4-hourly for two days	6.00 gm.
0.50 gm. three times daily for three days ...	4.50 gm.
0.25 gm. three times daily for five days ...	3.75 gm.
	38.25 gm.

This dosage is perhaps unnecessarily on the high side—see elsewhere in this paper—but it might be pointed out that dosages of 98 gm. in 14 days (Rappaport [7]), 80 gm. in 14 days (Stryker [8]) and 101 gm. (Perreau and Bayle [9]) have been given without serious toxic effects.

D. Duration of Stay in Hospital

From an administrative and financial viewpoint it is important to establish whether treatment with Chloromycetin reduces the average stay in hospital. We calculated the mean number of days spent in hospital by all cases, including those who died and had relapses, with the following results:

TABLE 2

	Days Spent in Hospital
Cases not treated with Chloromycetin	36·5, S.D. ± 22·3, S.E. ± 1·52
Cases treated with Chloromycetin	28·8, S.D. ± 20·8, S.E. ± 1·76

The difference is statistically significant.

French workers (Boquien *et al.*[10]) have found that the period spent in hospital by their Chloromycetin-treated group averaged 15 days and in their control group 27.3 days. In their treated group the total average dose of Chloromycetin was however some 30 gm., while in our series, as has been shown, the dosage was considerably less. The French series also included a large number of paratyphoid B cases, while ours were, as far as could be determined, mostly true typhoids. In the series of 289 cases of typhoid (not treated with Chloromycetin) reported by Armas Cruz *et al.*[1] the average duration of the disease was also recorded as 36.5 days (i.e. the same figure we reached).

E. Temperature Response

In the group not treated with Chloromycetin, with relapses and fatal cases excluded, the mean duration of fever was 22 days (S.D. ± 19, S.E. ± 1.47, coefficient of variation 86.4%). The 'normal' range of temperature was taken to be 97-99° F. The corresponding figure in the treated group was 6.3 days (S.D. ± 3.26, S.E. ± 0.35, coefficient of variation 51.6%).

The duration of the fever in the treated cases is rather longer than the 3.5 days reported by Woodward *et al.*[11, 12] and the 4.7 days reported by Boquien *et al.*[10] but Hirsowitz and Goldberg [4] found that their treated cases had a mean 6.5 days of fever, while their untreated cases were pyrexial for an average period of 12.8 days. They also excluded fatal and relapsed cases.

F. Relapses

Unfortunately, we could not determine for how long patients had been ill before being admitted to hospital. Relapsed cases were, thefore, defined as those cases which relapsed while in hospital. Death in relapse counted as a relapsed case when calculating relapse rates.

TABLE 3: RELAPSES IN CASES NOT TREATED WITH CHLOROMYCETIN

	Total Cases	*Death with Preliminary Attack*	*Relapses*	*Death with Relapse*	*Relapse Rate %*
Europeans ..	27	1	—	—	—
Native ..	185	35	11	1	6·5 ± 1·8
Indian ..	3	—	—	—	—
All races ..	215	36	11	1	5·6 ± 1·6

TABLE 4: RELAPSES IN CASES TREATED WITH CHLOROMYCETIN

	Total Cases	*Death with Preliminary Attack*	*Relapses*	*Death with Relapse*	*Relapse Rate %*
European	13	1	1	—	7·7 ± 7·4
Native ..	117	15	8	1	7·7 ± 2·5
Coloured	3	—	1	—	33·3 ± 27·2
Indian ..	6	—	1	—	16·7 ± 15·2
All races ..	139	16	11	1	8·6 ± 2·4

Comparing the relapse rate for natives in the two groups, the figure for those treated with Chloromycetin is higher, but the difference is not statistically significant. The percentage of relapses found in this series is lower than those reported elsewhere in the literature. Thus, for cases treated with Chloromycetin, Smadel *et al.*[13] reports a relapse rate of 13.6% (44 cases); Boquien *et al.*[10] a rate of 11.7% (60 cases); Perreau and Bayle [9] a rate of 13.0% (54 cases); El Ramli [11] a rate of 20.5% (200 cases) and Medina *et al.*[13] a rate of 18.0% (61 cases) and for cases not treated with Chloromycetin Boquien *et al.*[10] give a relapse rate of 6.7% (60 cases); El Ramli [16] a rate of 13.9% (122 cases) and Hirsowitz and Goldberg [4] a rate of 28.6% (21 cases).

It appears that in general, unless the treatment is adequate, relapses are more common in Chloromycetin-treated than Chloromycetin-untreated cases, and the occurrence of relapses in Chloromycetin-treated cases is bound up with:

1. Total dosage of Chloromycetin given, and
2. Duration of Chloromycetin therapy.

With regard to the first, Medina *et al.*[15] have shown that for children up to 11 years of age most relapses occur with a total dosage of under 15 gm. and for individuals over this age the bulk of relapses occur with a total dosage of less than 18 gm. With regard to the second (time) factor, Woodward, Smadel *et al.*[11, 12] have shown that most relapses occur when the treatment is given over a shorter period than nine days.

Pfeiffer [6] has pointed out that relapses even occur later than the tenth day after the cessation of Chloromycetin therapy. El Ramli [11] gives a mean apyrexial period before relapses in treated cases of 15.5 days compared with 4.4 days in untreated cases. Pfeiffer comments that, as a result, the average duration of time spent by a typhoid patient in an isolation hospital will be in the vicinity of four weeks, provided there are no complications, which is considerably less than the pre-Chloromycetin era when the disease lasted about six weeks. It is interesting how these estimates agree with our figures of 28.8 days for patients treated with Chloromycetin, and 36.5 days for the controls.

G. Complications

In our series of cases not treated with Chloromycetin 20 cases or 9.3 ± 1.9% developed complications which were noted in the records. How many were not noted cannot be determined but the specific complications recorded were breast abscess and basal pneumonia, perforation (died), pyelitis and typhoid spine, bronchitis, abortion, pleurisy, ulcerative ileitis (died), myocarditis (died), basal meningitis (died), anaemia, pharyngeal ulcer, parotitis, meningitis (died), cystitis, bronchopneumonia and thrombophlebitis. Five others were vaguely described as cardiac (died), toxaemia, fits and chest (two). More specific and accurate diagnoses would have been possible with better laboratory services and other diagnostic aids.

In the Chloromycetin-treated group two cases (1.4 ± 1%) were reported developing complications (both fatal). These were a perforation (in a European female aged 65) and a pulmonary catarrh with a probable perforation.

If we can assume that the records in the two groups are comparable, then there is a lower rate of occurrence in the Chloromycetin-treated group, which agrees with the findings reported elsewhere. Thus, Boquien *et al.*[10] reported 15% of his treated cases (60 in number) and 30% of controls (also 60 in number) as developing complications. El Ramli [11] reported 8.5% of complications among his 200 treated cases, and Armas Cruz [1] reported 43.6% among his 289 cases not treated with Chloromycetin.

H. Deaths

The number of deaths amongst Chloromycetin-treated and untreated patients, the time where they occurred after admission to hospital, and the case mortality rates are given in Table 5.

The standard error has been calculated for the case mortality rates, expressed in percentages.

Neither in the Europeans, the Natives, nor in the total group are the differences in this series statistically significant. This does not mean that Chloromycetin does not save more lives, but purely from a statistical point of view, the difference may still be due to chance. El Ramli [16] reported a case mortality rate of 6.5% in his 200 Chloromycetin-treated cases, and Armas Cruz *et al.*[1] reported a mortality rate of 9.7% in his 289 cases admitted to hospital in the pre-Chloromycetin era.

TABLE 5: TYPHOID PATIENTS—CASE MORTALITIES

		Not Given Chloromycetin			
		European	*Native*	*Indian*	*All Races*
Days in hospital	0	None	21	None	21
	10	None	5	None	5
	20	1	3	None	4
	30	None	5	None	5
	40	None	None	None	None
	50	None	1	None	1
	60	None	None	None	None
	70	None	1	None	1
Total deaths ..		1	36	None	37
Total cases ..		27	185	3	215
Case mortality		3·7	19·5	None	17·2
Standard error		±3·6%	±2·9%	None	±2·6%

		Treated with Chloromycetin				
		European	*Native*	*Indian*	*Coloured*	*All races*
No. of days in hospital	0	None	11	None	None	11
	10	None	3	None	1	4
	20	None	None	None	None	None
	30	1	None	None	None	1
	80	None	1	None	None	1
Total deaths		1	15	None	1	17
Total cases ..		13	117	3	6	139
Case mortality		7·7	12·8	None	16·7	12·2
Standard error		±7·4%	±3·1%	None	±13·9%	±2·8%

It will be noticed in our series that a large proportion of the deaths occurred shortly after admission to hospital. In the Chloromycetin-treated cases the period in hospital before death occurred was, of course, fairly closely related to the dosage of Chloromycetin received and of the 38 patients who received less than 10 gm. of Chloromycetin total dosage there were 11 deaths (29%) compared with six deaths (7%) amongst the 84 patients who received 10 gm. or more.

We found no evidence of the early deaths (after 1-3 days) reported by Sédallion *et al.*[17] supposed to be related to an inital heavy 'loading' dose of Chloromycetin—in fact early deaths (under 5 days) were more frequent in our control group (35% of all deaths) than in our Chloromycetin-treated group (24% of all deaths).

As however there seems to be no special advantage to be gained by giving a large 'loading dose' it might be safer if it were not used.

I. Carriers

According to Woodward *et al.*[12] their patients treated with Chloromycetin did not become chronic carriers. Their average dosage was 23.4 gm. over 9.2. days. Good and Mackenzie [18] reported that of six cases treated with 19-22 gm. over eight days the organisms persisted in three cases; Douglas [19] reported that two out of three cases who were

well treated excreted *S.typhi* in the urine and faeces for six months and 11 months (at time of writing) respectively, and Edge [20] found the organisms persisting in the faeces of eight out of 16 who were given an average dosage of 28.3 gm. for 9.5 days. We cannot add any additional information. In well-run isolation hospitals in South Africa, typhoid patients are not discharged until their excreta are free from *S.typhi*, but in many areas, where there are no laboratories, patients are discharged when they have recovered clinically, as there are no or limited facilities for bacteriological examinations of their stools and urine.

In a recent outbreak of typhoid at Mazeppa Bay, three cases followed on the visit of a white boy aged 11 years, who had been discharged from the Johannesburg Isolation Hospital on the thirty-fifth day after three successive negative stools and urines. His excreta could not be bacteriologically examined at Mazeppa Bay, but the presumption evidence is that he became a carrier after treatment with Chloromycetin (Parker [21]).

It has been frequently reported that Chloromycetin is of little value in the treatment of the carrier state—we need only refer to the reports of Rumball and Moore [22] —one failure. Collins and Finland [23]—one failure, Minkenhof [24]—four failures, Woodward *et al.*[12]—four failures, and Stryker [8]—one failure.

DISCUSSION

While it is clear that the use of Chloromycetin has decreased the number of days spent by each patient in hospital, and that the disease is more rapidly brought under control, it would appear that relapses are more common under Chloromycetin treatment as practised in South Africa in 1950, than in those cases not so treated. Heavier dosage over a longer period would probably decrease the incidence of such relapses. Whether country-wide death rates have been reduced has not been established. In this connexion it should be noted that many Native patients are brought to hospital in a moribund condition, under which circumstances it would be unreasonable to expect the exhibition of Chloromycetin to work miracles.

The limited evidence available suggests that Chloromycetin does not sterilize carriers or prevent the carrier state from developing. Detailed bacteriological studies in this connexion are urgently needed and could well be undertaken in this country.

Another point which should be investigated is whether the use of Chloromycetin interferes with the development of natural immunity to typhoid fever. Woodward *et al.*[12] stated that the Chloromycetin therapy was suppressive in nature and that recovery depended on the development of an immunity. It would be disconcerting if patients treated with Chloromycetin were subsequently again to develop the disease as a result of a second infection. It might be necessary to give artificial immunization to patients who have been treated with Chloromycetin.

SUMMARY

1. A study is made of 215 cases or typhoid fever not treated with Chloromycetin, and of 139 cases treated with Chloromycetin. These cases were mostly Natives, and the records were collected from hospitals in various parts of the Union.

2. The case mortality, while lower in the Chloromycetin group than in the control group, was not statistically significantly decreased.

3. The Chloromycetin group spent an average of 28.8 days in hospital while the control group spent an average of 36.5 days. The difference between these figures is statistically significant.

4. The average dose of Chloromycetin in the present series was 14.6 gm.

5. Average days of fever for the Chloromycetin group was 6.3, against 22.0 for the control group.

6. There were more relapses in the Chloromycetin group than in the control group. This difference was not statistically significant.

7. There was a lower rate of complications in the Chloromycetin group than in the controls. This was statistically significant.

8. The question of carriers is discussed. There is neither evidence that Chloromycetin decreases the carrier rate, nor that it cures chronic carriers.

Our thanks are extended to the Secretary for Health (Dr. G. W. Gale) and the Deputy Chief Health Officer, Tzaneen (Dr. D. H. S. Annecke) for the facilities made available to us and the encouragement given. The assistance rendered by Deputy Chief Health Officers, Medical Officers of Health, and Medical Superintendents of various hospitals, Provincial and Mission, from whom records were obtained, is also greatly appreciated.

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VAN DIE REDAKSIE

ROOK EN LONGKANKER

Gedurende die afgelope jare is belangstelling in die etiologie van longkanker opgewek deur die taamlik deeglike statistiese studies, waaroor verslae uit die Ou en die Nuwe Wêreld ontvang is.[1, 2] Hoewel die gevolgtrekkings nie dogmaties is nie, skyn dit asof daar sterk getuienis is dat daar 'n egte verband tussen die rook van sigarette en die ontstaan van longkanker mag wees. By die beoordeling van die toestand, ontstaan die moeilikheid gedeeltelik uit die feit dat daar op hierdie stadium slegs 'n korrelasie (al is dit ook 'n hoë en betekenisvolle) is tussen die rook van sigarette en longkanker. Daar moet nog bewys word dat hierdie twee sake, wat met mekaar in korrelasie verkeer, ook in oorsaaklike verband met mekaar staan.

Slegs deur die vindingrykheid van die statistikus kan baie maal die perke van gevolgtrekkings wat op syfers rus bepaal word. Daar is al beweer dat die toename, elke seisoen, in die geboortesyfer van die Skandinawiese lande betekenisvol met die gereelde trek van ooievaars na daardie wêrelddeel gekorreleer kan word! Versigtigheid is dus nodig om nie al te geredelik die gesag van syfers te aanvaar nie.

Sekere feite het nogtans duidelik uitgekom uit die verskillende studies[3]:—

1. Die seldsaamheid van kanker by 'n manlike pasiënt, wat nie tenminste 'n middelmatige kwaai roker vir baie jare was nie;
2. Die veel groter gebruik van sigarette onder pasiënte met longkanker, as onder andere van dieselfde ouderdom en inkomstegroep;
3. Die seksverspreiding van longkanker kom naasteby ooreen met die verhouding van langtermyn rookgewoontes tussen die twee sekse;
4. Die geweldige parallele toename in die verkoop van sigarette en die toename in hierdie vorm van kanker.

'n Reeks van 605 manlike gevalle van bronchogene kanker, met kontrolegroepe, is ontleed en drie onafhanklike studies is gemaak. Die gegewens was so eenvormig dat by elkeen dieselfde gevolgtrekkings toelaatbaar was.

Indien die rook van sigarette met 'n vorm van chroniese prikkeling gepaard gaan, wat tot die ontstaan van 'n gewas aanleiding kan gee, sou dit redelik wees om te vermoed dat kwaai rook en inaseming geassosieerde faktore is. Terwyl enersyds kwaai rook beskuldig is, is inaseming, andersyds, vrygespreek. Daar kan weinig twyfel bestaan dat groter akkuraatheid by vroeër diagnose die statistiese ontleding kompliseer en daar is gerugte dat verdere beoordeling van die gegewens wat reeds versamel is, aangedui het, dat daar 'n gewigtige korrelasie mag bestaan tussen sigaretrokers en die gebruik van sigaretaanstekers.

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EDITORIAL

SMOKING AND CANCER OF THE LUNG

In recent years interest in the aetiology of cancer of the lung has been stimulated by fairly thorough statistical studies which have been reported from the Old World and the New.[1, 2] Although the conclusions are not dogmatic, the evidence appears to be strongly suggestive that there may be a real link between cigarette smoking and the development of lung cancer. Part of the difficulty in assessing the situation arises from the fact that, at this stage, there is only a correlation (even though a high and significant one) between cigarette smoking and lung cancer. It still remains to be shown that these two things which are correlated with each other, are also connected causally.

Often the limits of what can be deduced from figures are determined only by the ingenuity of the statistician. It has been said that the seasonal increase in the birth rate in Scandinavian countries can be correlated significantly with the regular migration of storks to that part of the world! There is, therefore, need to be cautious about accepting too readily the authority of numbers.

Nevertheless, certain facts have emerged distinctly from the various studies[3]:

1. The rarity of cancer in a male patient who has not been at least a moderately heavy smoker for many years;
2. The much greater use of cigarettes amongst patients with pulmonary cancer than amongst other patients of comparable age and economic standing;
3. The sex distribution of pulmonary cancer corresponds roughly to the ratio of long-term smoking habits of the two sexes;
4. The tremendous parallel increase of the sale of cigarettes and of the increase in this form of cancer.

A series of 605 male cases of bronchogenic carcinoma was analysed, with control groups, three independent studies being carried out. The data were so uniform as to allow of similar conclusions in each of them.

If the smoking of cigarettes is associated with a form of chronic irritation which can lead to the development of a neoplasm, it would be reasonable to expect that heavy smoking and inhaling would be associated factors. While, on the one hand, heavy smoking has been incriminated, inhaling has, on the other hand, been exonerated. There can be little doubt that increased accuracy in earlier diagnosis complicates the statistical analysis and rumour has it that further interpretation of the data already collected has indicated that there may be a significant correlation, among the cigarette smokers, with the use of the cigarette lighter as opposed to matches. If, therefore, the postulate that there is a real connexion between

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in teenstelling met vuurhoutjies. Is die stelling derhalwe eg dat daar 'n wesenlike verband tussen die rook van sigarette en longkanker is, dan word dit al hoe moeiliker om die juiste karsinogeen vas te pen.

'n Verslag oor die situasie in Ysland mag veel lig op hierdie vraagstuk gedurende die loop van die volgende 10 tot 15 jaar werp. Longkanker kom selde voor in Ysland. Dungal [4] het 'n verspreidingssyfer van 0.6 persent in die loop van 2,000 lykskouings gerapporteer. Die gebruik van tabak egter, wat tot die uitbreek van die tweede wêreldoorlog baie gering was, neem tans vinnig toe. Ysland word derhalwe feitlik die laboratorium, waarin die beweerde verwekking van longkanker deur rook nou proefondervindelik getoets sal word. Dit is die gewone sigaret wat tot dusver beskuldig is. Dit sou ook belangwekkend wees om te weet of die Turkse sigaret met 'n minder mate van onheil as die Virginia smeul.

Hulle wat vertroosting en verskoning vir hul gewoontes soek, mag hulself troos dat nie iedereen daarmee akkoord gaan nie, dat kwaai rook met die ontwikkeling van longkanker verbind is en dat die eenvoudige pyp, sowel as die meer aristokratiese sigaar, sover nognie as so 'n onheilspellende voorteken beskou is nie.

4. Dungal, N. (1950): Lancet, **2**, 245.

cigarette smoking and lung cancer is a sound one, it becomes more and more difficult to pin down the actual carcinogen.

A report of the situation in Iceland may throw much light on the problem in the course of the next 10 or 15 years. Lung cancer in Iceland is rare. Dungal [4] reported the incidence of 0.6 per cent in the course of 2,000 autopsies. The use of tobacco, however, which was very little until the beginning of World War II, is now increasing rapidly. Iceland, therefore, virtually becomes the laboratory in which the experiment on the alleged production of lung cancer by smoking will now be carried out.

It is the vulgar cigarette which has so far been incriminated. It would also be interesting to know whether the Turkish cigarette smoulders with a less sinister smoke than does the Virginia.

Those who seek comfort and excuse for their habits may console themselves that not everyone is agreed that heavy smoking is associated with the development of lung cancer and that the homely pipe as well as the more aristocratic cigar have so far not been regarded as of such evil omen.

4. Dungal, N. (1950): Lancet, **2**, 245.

MACULAR CHANGES IN RIFT VALLEY FEVER

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Kimberley

The condition of macular oedema is a very interesting one. The variety to be discussed below will be labelled as central serous retinopathy following Duke Elder,[11] though there have been numerous other terms for the condition in the literature. Described by Von Graefe [22] in 1866 as central recurrent retinitis, the term used also by Fuchs [18]; Duggan [12] nevertheless claims that the first description was by Asayuma,[1] *et al* in 1898 as retinitis centralis. Other terms used, have been retinitis centralis annularis (Kraupa [28]); pre-retinal oedema (Guist [24]; Gissy [20]); idiopathic flat detachment of the macula (Walsh and Sloan [34]); central angiospastic retinitis (Horniker [25]); capillaritis (Bailliart [2]); central angiospastic retinopathy (Gifford and Marquardt [19]); choroiditis centralis serosa (Riehm [31]), retinal capillarosis (Cattanio [9]), angioneurotic macular degeneration (Candian [8]), choroidosis centralis serosa (Duggan [12]). Allied conditions are the chorio-retinitis centralis serosa of Japanese writers (Asayuma, *et al* [1]), Junius' juvenile exudative macular retinitis, Group 5 of Coat's disease, disciform degeneration of the macula, (Senile macular exudative choroiditis).

One reason for this variety of terms is the debatable question of the origin of the oedema. The one school of thought, which one might call the retinal school, considers that the exudation has occurred from the retinal blood vessels (Horniker,[25] Gifford and Marquardt,[19] Cattanio,[9] Candian,[8] Bailliart,[2] etc.); and the other school favours the origin from the choroidal circulation (The Japanese school,[1] Walsh and Sloan,[34] Duggan,[12]). For a full discussion Duggan's articles can be consulted. Gifford and Marquardt have stressed the angio-spastic element, but Greeves [23] in supporting the choroidal hypothesis, does not believe this exists as a clinical entity.

Here then is a characteristic oedema of the macular area, and limited to this area, which occurs especially in young adult males. There are no signs of inflammation, the patients are not hypertensive subjects and without general evidence of disease except possibly vasomotor lability in the extremities.

Early there is a clinical loss of retinal transparency. A faint circular light reflex only may be visible. Later an impression of a greyish ring enclosing a slight reddish centre, and later, glistening spots and faint lines, radiating from the fovea, appear. The area is usually less but sometimes equal to the size of the disc. Recently Goldman [21] stressed the importance of the slit lamp in the differentiation of doubtful cases from retrobulbar retinitis.

Aetiologically speaking there are several conditions which may give rise to the same picture. Various investigators have at different times blamed syphilis (Von Graefe,[22] Fuchs [18]); tuberculosis (Guist,[24] Gissy,[20] Scuderi,[32] Bonnet *et al.*[5]) Toxic causes (Batten,[3] Walsh & Sloan,[34] Kiewe and Reh,[27] Bonnet *et al.*[5] Karpe and Wising [26] have described it following infectious mononucleosis), vaso-motor instability (Horniker,[25] Bailliart,[2] Gifford and Marquardt,[19] Lucic,[29] Goldmann [21]), allergic reaction (Bettman,[4] Pallares,[30] Candian [8]). Several cases

of retinal detachment even have been reported as presumably allergic, e.g. Balyeat.

PROGNOSIS

This is generally fairly good. The visual disturbances disappear entirely or otherwise there is left a small relative scotoma with practically normal vision; a small absolute scotoma may remain. There is a tendency to recurrence. It is to be noted that choroid disease tends to leave a pigmented scar which means permanent visual defect (as distinct from spasm of the smaller branches of the retinal artery). The oedema disappears in 2-3 months but may last for 7-8 months (Elwyn [15]). To my knowledge no case attributable to Rift Valley fever has yet been described.

RIFT VALLEY FEVER

This is an acute virus infection producing liver necrosis in sheep and cattle, readily transmissible to man in whom it produces a febrile reaction, often tending to relapse, associated with prostration, headache and muscular pains (Findlay [14]). It was first described by Daubney, *et al* [10] in 1931 in Kenya, but since, it has been described in Uganda, Southern Anglo-Egyptian Sudan, French Sudan, French Equatorial Africa. Recently the disease has apparently spread to South Africa. The responsible agent is a filterable virus, 23-35 mμ in size, transmissible to various laboratory animals and is carried by an arthropod vector, possibly a mosquito. It is easily transmitted by laboratory infection and to those performing post-mortems on infected cattle and sheep.

Symptomatology. The incubation period is 3-4 days. There is headache associated with pains in the back. The conjunctivae are injected, and photophobia is common. The fauces are reddened; there is occasionally epistaxis. Appetite is poor. Occasionally there is nausea. Vomiting is rare. The temperature rises to between 103°F and 105°F for two or three days and then rapidly falls. There may be a secondary rise of temperature of 101°F or 102°F about 24 hours later and rarely a third or even a fourth pyrexial period. Each subsequent rise is less severe than the previous one. The liver and spleen are not enlarged. The urine is highly coloured. The pulse corresponds with the temperature for the first two or three days and then becomes slow. There is a primary leucocytosis followed by a leucopenia.

Diagnosis. The disease is suspected if a febrile reaction in man is associated with a febrile disease in cattle and sheep. In the febrile stage intra-peritonal injection of the infected serum into mice produces death in 48-72 hours with characteristic liver necrosis. After recovery, the CFT of Broom and Findlay [7] can be used or virucidal immune bodies can be demonstrated in the serum, and this is demonstrable for at least eight years (Findlay [17]). It may therefore be of interest to describe five cases of macular exudates and one case of retinal detachment recently seen in my practice. All of these have been proved serologically.

CASE HISTORIES

Case 1. Dr. S., a school teacher and author, aged 34 (with no history of contact with animals) had a febrile disturbance diagnosed as an acute pharyngitis. After a fever lasting two days, he had another relapse two days later, lasting two days and showed symptoms of an acute sinusitis (confirmed by X-ray). One week later he noted a gap in his central vision. When seen (three weeks later) the left fundus showed a large macular swelling, about the size of the disc and of a yellowish colour. There was a small haemorrhage to the temporal side of the oedematous area (Fig. 1). Fields as in (Fig. 2) V.L. was CF at 1 M. The right eye was normal with V.R. 6/6. He saw another ophthalmologist who confirmed the above findings. He then saw Dr. Maurice Franks, who reported as follows: 'The pupil reactions were equal and active to light, consensually and to accommodation. Ocular movements were normal as was the tension. Vision of the left eye was limited to counting fingers eccentrically, there was an obvious large central scotoma. Ophthalmoscopic examination of the left eye showed that the vitreous was clear.

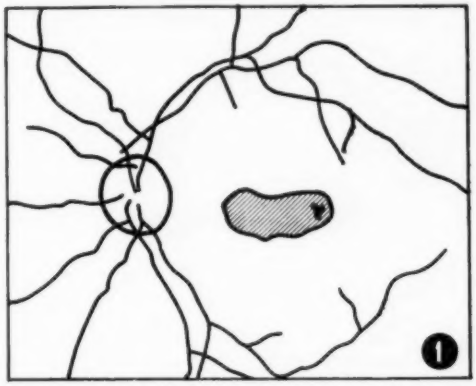

Fig. 1. Sketch of the fundus of Case 1. The shaded area represents a whitish mass at the left macula with a small haemorrhage at the temporal side of the mass.

The disc was normal but covering the left macula there was an elliptical white mass with the long axis horizontal. This mass was slightly raised and somewhat harder in texture than that seen in 'cotton-wool' patches associated with acute nephritis. The edges were clearly demarcated. A small capillary, arising from the inferior temporal branch of the central artery of the retina and going to the white mass, appeared to be thrombosed. The clinical appearance of this mass was suggestive of that commonly seen in central arterial embolism, but in this case it was localized to the macula and was still present a month to six weeks after the onset of the condition. It was thus apparent that the condition was more likely to be inflammatory and the possibility of the etiology being tick-bite fever or even Rift Valley fever was thought of.

The *diagnosis* of Rift Valley fever has now been confirmed.

The *prognosis* in regard to return of central vision in the left eye is not good and treatment will not be of much avail. Two months later, visual acuity had improved to 6/36, and the swelling had diminished considerably in size, without any pigmentary change. Needless to say, this diagnosis was totally unexpected by me, so with this in view, the following cases were re-investigated. In point of time, they had been seen previously to Case 1, and this paper was originally written attributing these cases to an unusual type of influenza.

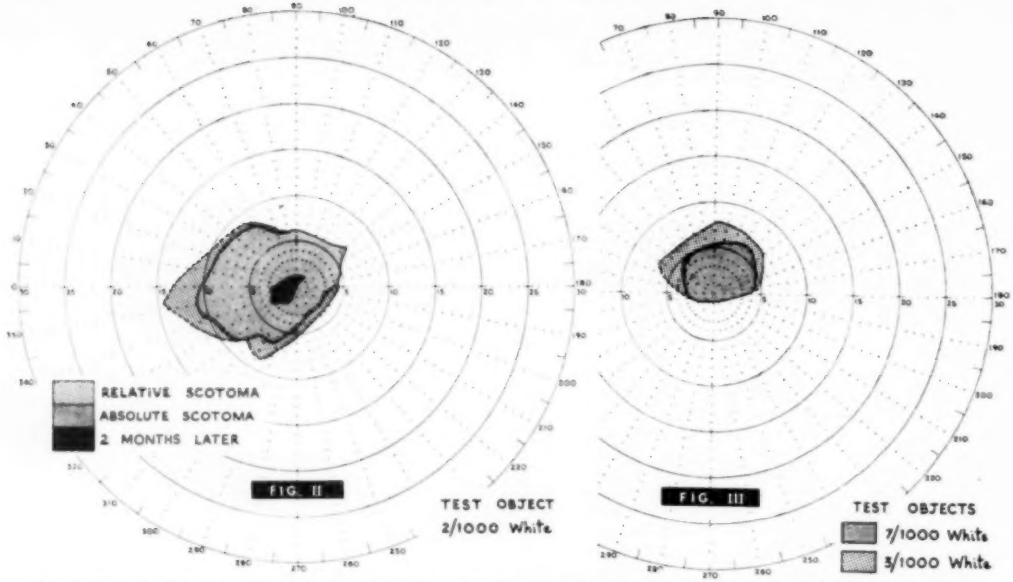

Fig. 2. Case 1 (left eye). C.F. at 1 M., on 28 May 1951. A large central absolute scotoma to 2/1,000 white with a smaller area of relative scotoma. The small central blacked-in area indicates improvement two months later.
Fig. 3. Case 2 (left eye). C.F. at 1 M., on 5 June 1951. Central scotoma with two different-sized test objects.

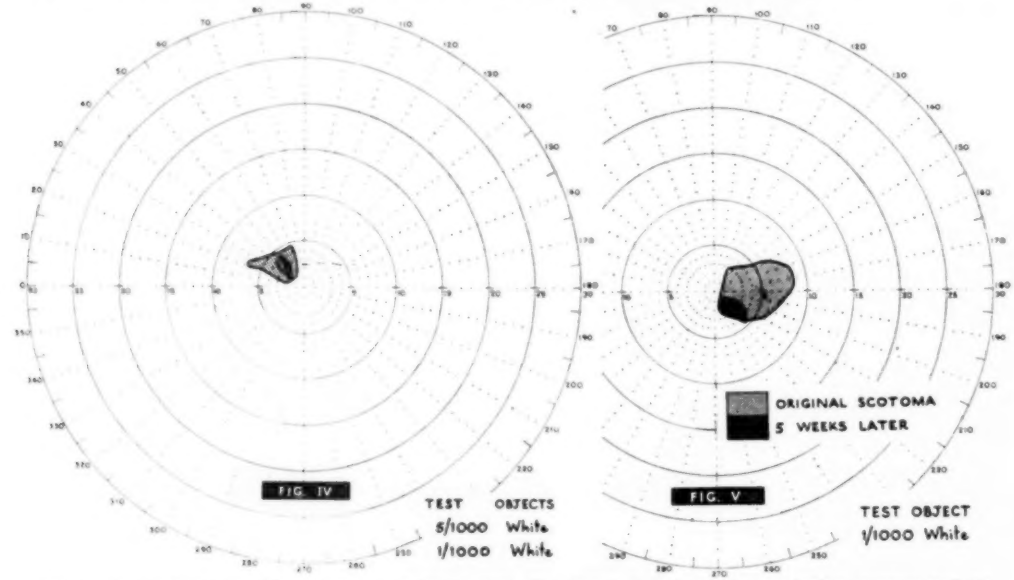

Fig. 4. Case 4 (left eye). V.L. = 6/18, on 15 March 1951. The densely-shaded area is an absolute scotoma and the stippled area is the relative scotoma.
Fig. 5. Case 5. Area of central scotoma. The smaller area shows how this has contracted in size in five weeks with improvement of V.A. from 6/18^{-1} to 6/9. This later improved to 6/6.

Case 2. Mr. B. aged 34, employed at a government research station. A number of cattle were found to have Rift Valley fever, and had been visited by the government vetinerary surgeon (Case 6). At the same time five of the staff developed a febrile disturbance (diagnosed as 'flu'). About one week later the patient noted that he could not see clearly out of the left eye. When seen by me about two months later V.R. was 6/6, right eye perfectly normal. V.L. was CF at 1 M, pupil reacted normally. The eye was 'white' and the fundus showed a large elevated yellowish area at the macula almost as large as in Case 1 and very similar in appearance (though without the haemorrhage). His central fields on the 1 M Bjerrum screen are as in Fig. 3. His doctor reported no evidence of organic disease. He was also diagnosed serologically as Rift Valley fever.

Case 3. Mr. C. aged 29, employed at the same station as Case 2 and also in contact with the same cattle. He had 'flu' at the same time as the other members of the staff. This lasted about one week and just after he had another attack of 'flu', milder in nature, lasting two or three days. The 'flu' consisted of a fever; weakness and sick feeling in the stomach. About three weeks later he noted a defect in vision of the left eye. When seen by me (about three months later) V.R. was 6/6 with a normal right eye. V.L. was 6/12 and at the macula was a very small exudate with a central haemorrhage. He too showed a small central scotoma on the Bjerrum screen. When seen two weeks later the haemorrhage had disappeared. The macula appeared clinically normal, but V.L. was still 6/12. His doctor could find no evidence of organic disease. Serological examination revealed a positive Rift Valley fever reaction.

Case 4. Mr. M., aged 28, employed at a government bag store; no direct contact with animals; had an attack of tonsillitis. Two weeks later he noted a 'blurred spot in the line of his vision'. V.R. was 6/6 with a normal right eye. V.L. was 6/18 and the fundus showed a large area of exudate just below the fovea. This was almost identical in appearance to Case 2. Two and a half weeks later this was becoming crenated at the edges and smaller in size, and one month later still had decreased to leave a small yellowish area at the periphery of the macula area. V.L. was only 6/12. He is otherwise healthy. Serologically examination revealed a positive Rift Valley fever reaction. Scotoma as in Fig. 4.

Case 5. Mr. R., aged 40, a diamond classifier, who had no contact with animals, had an attack of 'flu' and two or three days later noted 'fogginess' in the right eye. On examination about three weeks later he was found to have V.R. 6/18^{-1} (seen with head tilted) V.L. 6/6. Left eye was normal. Right eye showed the identical picture to Case 4 except that the macular exudate was in the upper macula periphery with a corresponding central scotoma. When seen six weeks later there was only a small area of exudate left and V.R. had improved to 6/9. On later examination, 12 weeks later the macula was normal to clinical examination and V.R. was 6/6. Serological examination revealed a positive Rift Valley fever reaction.

DISCUSSION

In a short space of time, I have therefore seen five cases, clinically similar, with macular exudates. Five are proven cases of Rift Valley fever (Table I). It appears possible therefore that Rift Valley fever may have a specific toxic effect on the macula. Whether the source of the central serous retinopathy is retinal or choroidal in nature is still debatable but in this respect I would like to mention the following:

Case 6. Mr. L. S., aged 50, government vetinerary surgeon, investigating suspected cases of Rift Valley fever in cattle, came to see me complaining of 'blurring of vision of sudden onset'

TABLE I: ANALYSIS OF CASES

Case	*Sex*	*Age*	*Occupation*	*Contact with Animals*	*Onset of Eye Symptoms after Pyrexial attack*	*Eye*	*Fundal Findings*	*When first seen*	*V.A.*	*V.A. of opposite eye*	*Progress*	*CFT for Rift Valley Fever*
Case 1: Dr. S.	M	34	Teacher	No	1 week	L	Large macular mass and small haemorrhage	3 weeks later	CF at 1.M.	6/6	V.A. improved to 6/36. Small mass still present	Positive
Case 2: Dr. B.	M	34	At Govt. Research Station	Yes	1 week	L	Large macular mass	2 months later	CF at 1.M.	6/6	Unchanged	Positive
Case 3: Dr. C.	M	29	At Govt. Research Station	Yes	3 weeks	L	Very small macular exudate with central haem.	2 months later	6/12	6/6	V.A. unchanged though macula clinically normal	Positive
Case 4: Dr. M.	M	28	At bagstore	No	2 weeks	L	Large paramacular mass	2 weeks later	6/18	6/6	V.A. improved to 6/12. Mass much smaller	Positive
Case 5: Dr. R.	M	40	Diamond classifier	No	2–3 days	R	Large paramacular mass	3 weeks later	6/18-^{1}	6/6	V.A. improved to 6/6. Macula clinically normal	Positive
Case 6: Dr. S.	M	50	Veterinary surgeon	Yes	Same time	R	Retinal Detachment	At the time of the pyrexia	CF at 1.M.	6/9-^{2} (with glasses)	In spite of operations, deterioration to PL.	Positive

in right eye. No history of injury. V.R. was CF at 1 M; with glasses was 6/9 -2. V.L. was CF at 1 M with glasses 6/6 -2. He was a medium-grade myope

(−4.50 D sph. R.E. / −3.00 D sph. L.E.)
(−0.75 D cyl. axis vertical / −0.50 D cyl. axis vertical)

The left eye appeared normal. The right eye had numerous floaters and a vitreous haze. He was sent to bed and while in bed developed 'flu', as diagnosed by his doctor. One week later the vitreous had cleared sufficiently for a diagnosis of spontaneous retinal detachment to be made. He was referred to a Johannesburg ophthalmologist, who performed two diathermy operations to seal off a large upper horseshoe hole and a number of small peripheral retinal dialyses below. He was proved to be serologically a case of Rift Valley fever. Several diathermy operations have been performed without success.

According to J. Pallares [30] many cases of central serous retinopathy are allergic in origin resulting from small doses of bacterial protein from some infected focus elsewhere in the body coming into contact with a previous sensitized macula (previous contact with the toxin). The exhibition of antihistamines combined with calcium cleared up the condition rapidly. Bettman's [4] case recovered on daily injections of epinephrine in oil. Accordingly, therefore, three cases were put on antihistamines and calcium but without marked success. The one case treated with aureomycin has shown the worst response of all.

SUMMARY

1. Five cases of central serous retinopathy have been discussed in otherwise healthy young men.

2. The fundus picture in at least four of these has been almost identical.

3. It is suggested that Rift Valley fever apparently is a cause of central serous retinopathy and it may also be a cause of a retinal detachment.

4. The prognosis of this type of case may not be as good as is generally stated.

I wish to thank Drs. S. Perel, N. Weinberg, D. Stephens and R. Nel, for their co-operation in investigating the cases referred to me. Also to Dr. M. Franks for the original diagnosis and Dr. J. Gear's assistance in getting me access to the journals available. Unfortunately the German literature was not available and had to be quoted in abstract form.

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RIFT VALLEY FEVER IN MAN

COMPLICATED BY RETINAL CHANGES AND LOSS OF VISION

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Human infection with the virus of Rift Valley fever was first reported in 1930 when Daubney, Hudson and Garnham [1] described its occurrence in four Europeans engaged in an investigation of an extensive epizootic in sheep in the Rift Valley in East Africa. Since then it has been shown to be fairly widespread in East and Central Africa. Schwentker and Rivers [6] noted that its manifestations, as an acute febrile illness in over two hundred cases known to have occurred in British East Africa, were usually of such a mild character that no untoward sequelae were observed. That this is not invariably true was demonstrated by Schwentker and Rivers [6] when one of their pathologists contracted the infection whilst working with the virus in their laboratory. A popliteal vein thrombosis occurred on the tenth day of the illness, pulmonary embolic phenomena occurred on the twentieth, twenty-sixth and thirty-fourth day, and on the forty-fifth day sudden collapse and death. Post-mortem examination revealed extensive thrombus formation in the inferior vena cava and the hepatic venous radicles and with extension upwards almost to the heart. Embolus was found in the pulmonary artery. The liver was normal.

The purpose of this communication is to report another, though less formidable, but hitherto undescribed sequela of Rift Valley fever in a human, viz. loss of central vision commencing on the sixth day of the illness and to give an account of the fundoscopic appearances. Although Daubney, Hudson and Garnham [1] stated that one of their

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four cases complained of headache and defective vision for some weeks after the illness, no mention is made as to the nature of the visual defect or as to whether it was temporary or permanent.

A 38-year-old schoolmaster was examined on 17 June 1951 for the complaint of disturbance of vision of the left eye of 6 weeks' duration. He stated that he had taken ill suddenly in Kimberley on 3 May 1951 with a rigor, generalized aching of the body, severe pain behind the eyeballs and fever of 103° F. After 48 hours the symptoms and the fever abated completely. He felt quite normal for the following two days, after which time he had a recurrence of fever, pain behind the eyeballs and generalized body pains. This continued for a further two days, when symptoms once more disappeared. Six days after the onset of the illness he noticed blurring of vision of the left eye, which had persisted to the present time. Apart from a feeling of slight debility he had no other complaints. Treatment during the febrile stage consisted of sulpha drugs and Penicillin.

Examination revealed an essentially normal condition of the skin, mucous membranes, cardiovascular and respiratory systems. No abnormality was detected on abdominal palpation. Peripheral lymph glands were not palpable. Neurological examination showed no abnormality, except for a difficulty in being able to elicit the patellar jerks. All other reflexes were present, normal and equal. No sensory changes were noted.

Examination of the right eye showed normal ocular movements, tension, vision and fundoscopic appearances. The left eye showed normal ocular movements and tension, but vision was limited to counting fingers eccentrically and there was an obvious large central scotoma. Funduscopic examination showed a clear vitreous. The disc was normal, but covering the left macula there was a dense white elliptical mass with its long axis horizontal. The mass was slightly raised and harder in texture than that seen in the 'cotton wool' patches associated with acute nephritis. The edges were clearly demarcated. A small capillary, arising from the inferior temporal branch of the central artery of the retina and going to the white mass, appeared to be thrombosed. The clinical appearance of this mass was suggestive of that commonly seen in central arterial embolism, but in this case it was localized to the macula and was still present 6 weeks after the onset of the condition, so that its aetiology was more likely to be of an inflammatory nature.

The features of the case, then, were a short febrile illness of sudden onset, retro-orbital pain and a saddle-back Dengue type of pyrexia with retinal changes as an early complication. Laboratory studies to elucidate the nature of this illness were then instituted:

25 June 1951 SEROLOGICAL TESTS

The Macnab-Lewin modified Ide screen test yielded a *negative* result.
The Eagle flocculation test yielded a *negative* result.
The standard Kahn test yielded a *negative* result.
The Paul-Bunnell test (Barrett's modification) yielded a *negative* result.

Serum absorbed by	*Titre*
Guinea-pig kidney	0
Ox cells	10
Unabsorbed	20

(Serum submitted to the South African Institute of Medical Research for Rift Valley fever and Q fever.)

25 June 1951 URINE

Appearance: Clear yellow
Reaction: Alkaline
Protein: Absent
Sugar: Absent
Deposit: Macroscopic—nil

Microscopic examination of the centrifugate showed a very occasional polymorphonuclear leucocyte averaging less than one per high power field. Casts, crystals, erythrocytes and inflammatory cells were not seen.

AGGLUTINATION REACTIONS

Antigen	*Titre*							
	25	50	100	200	400	800	1,600	3,200
B. typhosus O ..	—			—				
B. typhosus H ..	—		—					
B. paratyphosus A	—		—					
B. paratyphosus B	—		—					
B. paratyphosus C	—		—					
*B. proteus OX*19		—		—				
*B. proteus OX*2 ..		—		—				
B. proteus OXK ..		—		—				
Br. abortus ..	—	—	—	—	—	—	—	—
Br. melitensis ..	—	—	—	—	—	—	—	—

Cold agglutination titre: 0.

REPORT ON ONE SPECIMEN OF SERUM

COMPLEMENT FIXATION TESTS (27 JUNE 1951.)

Antigen	*Result*	*End Titre*
R. prowazeki (epidemic typhus) ..	Negative	
R. mooseri (murine typhus)	Negative	
R. rickettsi (tick-bite fever)	Negative	
R. akari (rickettsial pox)	Negative	
R. burneti (Q fever)	Negative	
Rift Valley fever virus	Positive	1:50x

This result indicates that this patient has had Rift Valley fever.

Since specific neutralizing antibodies for Rift Valley fever virus were demonstrated in this patient's serum, and as the retinal changes had appeared 6 days after the onset of the infection, it seemed not unreasonable to attribute these changes to the virus infection. In order to determine whether the changes were reversible, an effort was made to institute Aureomycin therapy for a period of a week. A re-examination of the fundus was made on 13 July 1951, i.e. 4 weeks after the first examination, and 10 weeks after the onset of the visual disturbance. No material change in the appearance of the size or shape of the white elliptical mass over the macula was observed, and the central scotoma remained.

DISCUSSION

It was not possible to determine the original source of infection in this patient with any exactitude. He had visited a farm in the Christiana district 4 weeks before the onset of his illness, at which time he stated that there

was some sickness amongst cattle. An incubation period of such long duration, however, is far in excess of the 4- to 6-day period which is customarily accepted (Theiler [7]). The likelihood that he may nevertheless have contracted the infection in Kimberley remains, a possible mode of spread being an infected domestic animal or a bloodsucking arthropod, the latter infecting the man secondarily. That evidence for such a cycle exists was demonstrated by Daubney, Hudson and Garnham [1] in the Kenya epizootic when they observed that in an infected region sheep could be protected by screening at night and that removal of the herd to the highlands, which were mosquito-free, brought about a cessation of the epidemic. The virus has been shown to survive for several days in a variety of mosquitoes. In studies in the Bwamba forest conducted by the Yellow Fever Research Institute in Uganda, the virus of Rift Valley fever was isolated on several occasions from wild-caught mosquitoes belonging to the species *Eretmopodites chrysogaster*. It is probable that domestic animals are infected by some blood-sucking arthropod, and that man as a rule seems to become secondarily infected during the course of an epizootic. The finding of the virus in wild-caught mosquitoes of the Bwamba forest is highly suggestive of some virus cycle in wild animals (Theiler [7]).

Experimentally, a large variety of animals has been shown to be susceptible when inoculated with the virus. Theiler[7] states that all monkeys, sheep, goats and cattle are readily infected; other susceptible animals are the mouse, ferret, hamster, white rat, various species of wild European and African rodents, and possibly the rabbit. Animals not susceptible are the horse, pig, guinea-pig, chicken, canary and pigeon. Mice are particularly susceptible, death occurring usually within 2 or 3 days after inoculation.

Whilst the virus is normally viscerotropic in its effect on animals, lesions being found characteristically in the liver in the form of widespread necrosis, such is curiously not the case in human infections. In the autopsy report of a human case described by Schwentker and Rivers, the liver was found normal. In the patient here reported the lesion was confined to the retina. The question therefore arises whether a modified strain of the virus was not involved in this case. That a strain other than viscerotropic type may exist was amply demonstrated by Mackenzie and Findlay [3] and Mackenzie, Findlay and Stern,[4] who produced a neurotropic strain by inoculating mice intracerebrally immediately after an intraperitoneal injection of immune serum. By serial passage in such passively immunized animals the virus lost, to a considerable extent, its viscerotropic affinities, so that it no longer produced death due to liver lesions when inoculated subcutaneously. Inoculated intracerebrally, the modified virus produced a fatal meningo-encephalitis in mice and monkeys. On subcutaneous inoculation into adult sheep, the neurotropic virus caused no reaction. The animals, however, developed antibodies and were resistant to an inoculation of unmodified virus. It is interesting to note that in lambs less than 4 weeks of age the subcutaneous inoculation of the neurotropic virus produced a fatal encephalitis. That no attenuation occurs for man by prolonged passage in mice is shown by the accidental infection of a worker with virus which had undergone at least 300 passages in these animals (Sabin and Blumberg).

SUMMARY

A case of Rift Valley fever in a human subject is presented.

A sequela of retinal changes with loss of central vision, hitherto undescribed in the literature, is reported.

The possible mode of infection, on the basis of reported experimental infections, is discussed.

The question of a neurotropic strain in human infection is raised.

I am indebted to Dr. J. H. S. Gear, of the Rickettsial and Virus Diseases Laboratory, South African Institute for Medical Research, for the blood studies which established the diagnosis and to Dr. W. Lewin, of Medical Centre, Johannesburg, for the other laboratory studies. I have to thank Dr. Maurice Franks and Dr. A. Jokl, of Lister Building, Johannesburg, for their detailed descriptions of the funduscopic appearances.

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A RACK FOR RETINOSCOPY

S. ETZINE, M.B., CH.B., D.O.M.S. (ENG.)
General Hospital, Johannesburg

'I judge the art of surgery the more certain', wrote Montaigne in an essay *Of Surgeons and Physicians*, 'for it seeth and handleth what it doth and therein is less conjecture and divination, whereas Physicians have no *speculum matricis* to discover our brain, our lungs and our liver unto them.' The ophthalmic surgeon is fortunate in having the technique of retinoscopy to remove much of the conjecture and divination from the study of the refraction.

Sir William Bowman, in 1859, is reputed to have been the first to record the variable displacement of light reflected from the retina in different states of the refraction. This forms the basis of retinoscopy. He did not, however, grasp the general application of the observation

he had made. Curiously enough, it was in Africa that retinoscopy originated. Cuignet, a French military surgeon attached to a North African battalion, was faced with a problem common to army doctors in all countries, that of the malingerer. An objective measure of the refraction was clearly desirable in patients whose veracity might be in question. The ophthalmoscope had been invented in about 1850 by Helmholtz. Cuignet found that if he illuminated the pupil of an eye with this instrument, the displacement of the shadows bordering the illuminated zone varied with the state of the refraction. His first paper on the subject appeared in 1873. In the belief that the light reflected from the eye came from the cornea, he applied the term keratoscopy to the technique of the examination. Other ophthalmologists were quick to contest this opinion. A détente was brought to the controversy by the introduction of the non-committal term skiascopy or examination of shadows. This term is still used in France today to describe the technique known as retinoscopy in English-speaking countries. It was left to another French surgeon, Parent, to put the theory and practice of retinoscopy on a sound basis and to popularize the method. In 1880 he showed the advantage of using a plane mirror, of working at a distance of 1 metre and of interposing correcting glasses until a reversal of the movement of the shadows was obtained. He is considered the father of retinoscopy and in the circumstance his name is appropriate.

Retinoscopy Racks. A retinoscopy rack is a device designed to expedite the rate at which the lenses placed before the patient's eyes are changed. Numbers of these racks are in existence. Most of them are shaped like rules and consist of an equal number of convex and concave lenses arranged in increasing strengths. While the average case of lenses contains 60 different spherical glasses, it is manifestly difficult to arrange so large a number of lenses in linear form without making the instrument cumbersome. In practice, therefore, some lenses have to be left out. Most racks contain about 24 lenses ranging up to 12 dioptres at intervals of 1 dioptre or up to 6 dioptres at intervals of 0.5 dioptre. The writer feels that the disposition of the lenses in these racks is arbitrary and that the construction of a retinoscopy rack should be based upon a task analysis which should include a study of the frequency distribution of refractions. The following considerations merit attention.

1. *Weight of the Rack.* The rack is held at arm's length by the examiner. A heavy rack adds to the day's fatigue. The weight of the rack can be reduced by having fewer lenses, by using a light plastic material as a frame and by reducing the diameter of the lenses. Foster has shown that it is unnecessary to employ lenses larger than 6 mm. since the pupil seldom exceeds this size.

2. *Length of Rack.* A long rack is difficult to manipulate. The contours of the face may cause the rack to tilt, especially if it is long. The effect will be to make incident light fall obliquely on the lens. Such eccentric pencils of light are not brought to a point focus, but are refracted astigmatically. An error is introduced in this way, which is of appreciable dimensions in the case of lenses of higher power. These are therefore best omitted from a rack.

3. *Working Distance.* The rack held at arm's length is nearer to 60 cm. than the 100 cm. which is theoretically desirable. In Paris it was at one time the custom for the patient to hold and to move the rack in front of his own eyes while the surgeon sat at a distance of 1 metre. The writer's experience of patients in this country is that they lack the *savoir faire* of the Parisians and cannot be expected to co-operate in the examination in this way. The reduced working distance entailed by the use of the rack is no real disadvantage, as the observer soon learns the correction proper to his own working distance.

4. *Interval between Lenses.* The smaller the interval between the dioptric strength of successive lenses, the greater the accuracy of the instrument. Just as a rule which measures to the nearest quarter-inch is more accurate than a rule which measures only to the nearest half-inch, so a rack which has 0.25 D intervals will be more accurate than one in which the lenses succeed each other at 0.5 D or 1.0 D intervals.

5. *The Frequency Distribution of Refractions in Clinical Practice.* The frequency distribution of refractive states in unselected cases has been studied by a number of workers (Stenström). It seemed to the writer that the distribution might differ in clinical material, since patients with refractive errors would be expected to be seen more frequently at ophthalmic consultations than persons who were approximately emmetropic. Since the retinoscopy rack is essentially a clinical instrument, it was felt that the study of a consecutive group of clinical cases, in contrast to unselected material, would be of interest. The writer therefore undertook the analysis of the refractive states in 1,000 eyes examined consecutively in his consulting rooms. The patients were aged from 16 to 73 and included both males and females. Both eyes were included in the study. For the purpose of the analysis, the meridian which deviated most from the emmetropic was used. A histogram was constructed from these figures (Fig. 1). This did not confirm the supposition

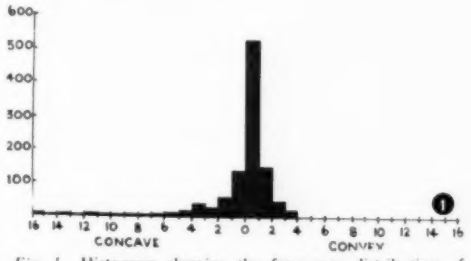

Fig. 1. Histogram showing the frequency distribution of refractions in 1,000 eyes (clinical material).

that it would differ markedly from one for an unselected series. The frequency distribution for the clinical cases studied corresponded closely to Stenström's figures for unselected refractions. More than half the cases fell between the range 0 to 1.0 D convex. Half of the remainder fell almost symmetrically about a range of 1.0 D on either side of the zone of maximum incidence.

It will be seen from the graphical representation that the frequency of refractions does not have a binomial distribution. The curve shows excess to the positive side and skewness to the myopic side.

The range of refractions is usually considered to be between −20.0 and +12.0 D. Analysis of the figures shows that 80% of the refractions in this series were concentrated between −1.50 and +2.0 D, i.e. over 3.5 D, while the remaining 20% were sparsely distributed over the remaining 28.5 D.

Author's Retinoscopy Rack. This was constructed on the basis of the above analysis. The lenses included are from 0 to +3.50 D at 0.25 D intervals. Allowing for a correction of −1.50 D, they cover the zone of maximum density and include 80% of the refractions encountered. The diameter of the lenses was as small as local conditions of manufacture would allow. A light plastic frame was used. The instrument was manufactured in Johannesburg by Basman, Berman and Woodward, and has proved useful in practice.

SUMMARY

A task analysis leads to the conclusion that the greatest utility of a retinoscopy rack is in covering the zone of maximum concentration of the refractions. Attempts to cover the whole range of refractions with this type of instrument are made at the expense of accuracy and convenience.

The writer's instrument is described.

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NEW PREPARATIONS AND APPLIANCES

Paediatric Chloromycetin Palmitate

The administration of Chloromycetin to infants and to children who will not swallow the 50 mg. capsule constituted, until recently, a practical difficulty on account of the extremely bitter taste of the antibiotic. Research on this problem resulted in the development of Chloromycetin Palmitate, an insoluble and, therefore, bitterless ester of Chloromycetin, which hydrolyses in the gastro-intestinal tract to release pure Chloromycetin.

Paediatric Chloromycetin Palmitate is a pleasant-tasting suspension of the palmitate. This form of Chloromycetin facilitates the administration of the antibiotic to children and to other patients unable to take capsular medication. Each 4 c.c. (1 teaspoonful) contains Chloromycetin palmitate equivalent to 125 mg. of Chloromycetin.

Indications: The wide anti-bacterial effectiveness distinguishing the clinical performance of Chloromycetin is equally characteristic of the activity of Paediatric Chloromycetin Palmitate. Clinical indications for the liquid preparation are the same as for the capsular form of Chloromycetin and the product is primarily intended for administration to children. Infections in which this antibiotic is effective include pertussis, gastro-enteritis, pneumonia (both bacterial and primary atypical), laryngo-tracheo-bronchitis, urinary tract infections as well as typhoid and paratyphoid fevers.

Dosage: For infants and children the suggested dosage of Chloromycetin is 75 mg. per kg. of body-weight daily, in divided doses, at intervals of four to six hours, reducing to 30 mg. per kg. daily. A daily dosage of 100 mg. per kg. of body-weight is suggested in severe infections including pneumonia and whooping-cough. In infantile gastro-enteritis a daily dosage of 150 mg. per kg. of body-weight is recommended.

In practice this means that *infants up to one year may be given one teaspoonful of* Paediatric Chloromycetin Palmitate *every four to six hours around the clock* depending on the patient's weight and clinical condition. *Children over one year may receive one to two teaspoonfuls every four to six hours.* After the initial stages these doses may be somewhat reduced.

(*Note*: The use of this product, as with other antibiotics, may permit a growth of non-susceptible organisms, particularly *Monilia albicans*.)

Package: Paediatric Chloromycetin Palmitate is supplied in 60 c.c. bottles, each teaspoonful (4 c.c.) containing the equivalent of 125 mg. Chloromycetin.

Manufactured by Parke, Davis & Company, and distributed by Lennon Limited, P.O. Box 8389, Johannesburg.

SOUTH AFRICAN MEDICAL AND DENTAL COUNCIL

Reports of Disciplinary Enquiries

[These reports have been amended in the interests of the anonymity of the colleagues concerned.—*Editor.*]

1. At the last meeting of the South African Medical and Dental Council, it considered the report of a Special Disciplinary Enquiry which held an inquiry into the conduct of Dr. K. M. The following was the charge preferred:

'That you, being a medical practitioner registered under the provisions of the Medical, Dental and Pharmacy Act 1928 as Amended, are guilty of improper conduct or disgraceful conduct or conduct which when regard is had to your profession, is improper or disgraceful, in that you were convicted in the Magistrate's Court on 2 February 1951, of (1) having wrongfully and unlawfully, in public or private, committed or been party to the commission of an act of gross indecency with another male person, a 10-year-old child; (2) having wrongfully and unlawfully, in public or in private, committed or been party to the commission of an act of gross indecency with another male person, a 10-year-old child, in respect of which conviction you were sentenced to one month's imprisonment with hard labour on each count, suspended for three years on condition that you are not again convicted of a similar offence during that period.'

The Disciplinary Committee found Dr. K. M. guilty of improper conduct and resolved that the penalty to be imposed upon him be that he be cautioned. The Council confirmed the finding of the Disciplinary Committee and of the penalty imposed.

2. At the last meeting of the Council, it considered a report by the Executive Committee of the Council on an inquiry which the Executive Committee held into the conduct of Dr. S. M. K.

The following was the charge preferred:

'That you, being a medical practitioner registered under the provisions of the Medical, Dental and Pharmacy Act,

1928 as amended, are guilty of improper conduct or disgraceful conduct or conduct which when regard is had to your profession, is improper or disgraceful in that on 4 February 1951, you granted, without qualification, a medical certificate of still-birth (Form B.M.D. 11) wherein you certified that on 4 February 1951, you attended during the birth of and examined the body of a female child, and that the said child was not born alive, whereas in truth and in fact you did not attend the birth of the said child, nor did you examine its body, nor could you truthfully certify that the said child was not born alive.'

The Executive Committee found Dr. S. M. K. guilty of conduct which when regard is had to his profession, is improper, and it resolved that he be cautioned. The Council confirmed the finding of the Executive Committee, and the penalty imposed.

PASSING EVENTS

Mr. C. A. R. Schulenburg (whose rooms are at 50 van Riebeek Medical Buildings, Schoeman Street, Pretoria) has changed his telephone number to 3-2841.

* * * *

The U.S. Food and Drug Administration has increased the expiry dates for Terramycin capsules, elixir and oral drops from a period of one year to three years; and ophthalmic ointment, ophthalmic solution, troches and topical ointment now expire in two years.

* * * *

Dr. S. Eisenhammer of Johannesburg has returned after a visit of six months to England and the United States for the purpose of doing post-graduate work.

* * * *

Prof. E. C. Crichton has retired from the Chair of Obstetrics and Gynaecology at the University of Cape Town and is now in private practice at 78 Queen Victoria Street, Cape Town. Telephone: 2-8006.

* * * *

Drs. Jack Abelsohn, N. C. Smiedt and P. S. Jenkin (the latter formerly Senior Assistant Anaesthetist at Groote Schuur Hospital) have entered into partnership and from 1 January 1952 will practice as specialist anaesthetists at 503 African Life Buildings, St. George's Street, Cape Town.
Telephones: 3-0483 and 2-7881.

Dr. R. Campbell Begg of Johannesburg has returned from a visit to Great Britain and the Continent including Greece and Turkey.

MOTOR CAR INSURANCE FOR MEMBERS OF THE MEDICAL ASSOCIATION

Members are reminded that it will be to their benefit to reply to the circular recently forwarded to them with a newsletter. Doctors using their cars for professional purposes can effect a considerable saving in the premium to be paid by communicating with Messrs. Edward Lumley and Sons, P.O. Box 4639, Cape Town, the Association's official insurers for this class of motor-car insurance.

AWARD FOR OUTSTANDING RESEARCH IN THE FIELD OF INFERTILITY

The American Society for the Study of Sterility announces the opening of the 1952 contest for the most outstanding contribution to the subject of infertility and sterility. The winner will receive a cash award of one thousand dollars, and the essay will appear on the programme of the 1952 meeting of the Society.

Essays submitted in this competition must be received not later than 1 March 1952. For full particulars concerning requirements of this competition, address The American Society for the Study of Sterility, 20 Magnolia Terrace, Springfield, Massachusetts, U.S.A.

THE BENEVOLENT FUND

The following contributions to the Benevolent Fund during October 1951, are gratefully acknowledged:

Votive Cards: *In Memory of*:

Miss L. Werge *by* Dr. and Mrs. A. W. Sichel.
Dr. J. A. Weir *by* Dr. E. Bruce, Mrs. W. L. Dobbie, Dr. and Mrs. W. Wilkie, Dr. and Mrs. P. S. Jenkin.
Mr. P. Hofmeyr *by* Dr. and Mrs. J. C. Gie, Mrs. A. Spratt.
Dr. B. Vivier *by* Dr. J. D. Wicht.
Dr. P. J. G. de Vos *by* Dr. and Mrs. L. R. Brumberg and Family, Dr. A. W. Sichel, Dr. and Mrs. L. M. Marchand, Dr. J. Black, Dr. H. Aneck-Hahn, Drs. Welsh, Lawrance and Lloyd-Wronsley, Dr. C. A. R. Schulenburg, Mr. and Mrs. Walter Webber and Family, E. M. Wright, Dr. and Mrs. A. H. Tonkin.
Mr. A. G. Shave *by* Dr. H. O. Hofmeyr.
Dr. Max Greenberg *by* Dr. A. W. Sichel.
Dr. C. C. P. Anning *by* Dr. A. W. Sichel.
Dr. Hugh Croudace *by* Dr. H. E. Brown.

Total Amount received from Votive Cards: 30 8 6

Services Rendered to:

Dr. H. A. Edwards *by* Drs. G. Dean and John Lee.
Dr. S. Osrin *by* Prof. F. Forman.
Dr. J. H. McLean *by* Dr. A. B. W. Ferreira.
Dr. A. Lawson *by* Dr. J. D. Joubert.
Dr. M. Sterne *by* Dr. Dommisse.
Mrs. Roos *by* Dr. H. G. Owen-Smith.
Dr. A. G. Sweetapple *by* Drs. J. W. Schabort, E. Samuel, M. Suzman, L. I. Braun and C. Adler.

Total Amount received from Services Rendered: 39 18 0

Donations:

Dr. G. Radford 5 5 0
Dr. C. H. Derksen 7 4 0
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The Medical Officers of Health (State Medicine) Group of the M.A.S.A. 10 10 0

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REVIEWS OF BOOKS

PRACTICAL ENDOCRINOLOGY

The Practice of Endocrinology. Edited by Raymond Greene, M.A., D.M., M.R.C.P. (Pp. 389 + xxiii, with 56 illustrations and 25 figures. 65s.) Published on behalf of The Practitioner by Eyre & Spottiswoode Limited. 1951.

Contents: 1. Introduction. 2. The Hypothalamus. 3. The Pituitary Gland. 4. The Adrenal Glands. 5. Diseases of Adaptation. 6. Sex and Reproduction. 7. The Thyroid Gland. 8. Carbohydrate Metabolism and Diabetes Mellitus. 9. Calcium Metabolism and the Parathyroid Glands. 10. The Thymus and Pineal Body. 11. Corpulence. Index.

Practitioner handbooks fulfil with unique distinction an important functon in contemporary medical writing. They are undoubtedly written so as to be a source of great usefulness to the general practitioner.

The first edition of this excellent manual was published only some three years ago, and the fact that a new edition is now available is a quiet but eloquent tribute to the great utility of this volume. The section dealing with *Corpulence* is particularly valuable and is characterized by a common sense which pervades the whole volume. The chapter on *Diseases of Adaptation* recognizes that this concept has now passed into the current knowledge and language of the general physician. The concept is explained very lucidly. Moreover, it is not surprising that a modern volume on endocrinology

virtually covers the most important fields of medical practice. In the case of this book, the task is done with skill and keeps the reader fully abreast of developments in theory as well as in treatment.

The volume is most attractively produced. The illustrations, especially the colour ones, are of a high order and there can be little doubt that the undergraduate student of medicine as well as the practising doctor will profit considerably from this book.

SCHOOL HEALTH SERVICES

Expert Committee on School Health Services: Report on the First Session. (Pp. 36. 2s.) World Health Organization Technical Report Series No. 30. Geneva: Palais des Nations. 1951.

Contents: 1. Introduction. 2. Reasons for Special Consideration of the School-age Group. 3. What can be done for the Health of the School-child. 4. Staff and Training required for a School Health Service. 5. Framework of Organization and Administration into which the School Health Services can be fitted. 6. Further Studies. 7. Conclusion.

This Report is up to the standard that can be expected from World Health Organization level. The time allotted to the Committee limited the report to an outline of basic policies and approaches. They stressed the great advantage to be derived from integrating the administration of the school health service with that of the general community health programme. While the service will be sharply affected by problems of social and medical organization, and by the economics of the various countries and areas of the world, it was stressed that the basic needs of each child to attain his own maximum status, was the same, irrespective of race, climate, geographical location, the particular school attended, or the stage of technical development of country and community.

It was further emphasized that most lasting improvement in public and individual health will come through changing attitudes and developing understanding; in short, through education. As much as possible should be done by the people, by their co-operative action, and as little as possible by unexplained intervention.

This Report will be of great interest to educational and health authorities. It is presented in a clear manner, and it is a pleasure to read it.

THE BRITISH ENCYCLOPAEDIA OF MEDICAL PRACTICE

The British Encyclopaedia of Medical Practice. Vol. 6. Under the General Editorship of the Rt. Hon. Lord Horder, *G.C.V.O.*, M.D., F.R.C.P. (Pp. 680 + xv + Index, with 123 figures. 66s. per volume. 2nd edition.) Butterworth & Co. (Africa) Limited, 1 Lincoln's Court, Masonic Grove, Durban. 1951.

Contents: 1. Gastritis. 2. German Measles. 3. Glanders. 4. Glandular Fever. 5. Glaucoma. 6. Glycogen Disease. 7. Glycosuria. 8. Gonorrhoea. 9. Gout. 10. Granuloma Inguinale. 11. Guinea-Worm Disease. 12. Haematemesis. 13. Haematuria. 14. Haemochromatosis. 15. Haemoglobinuria. 16. Haemophilia. 17. Haemoptysis. 18. Haemorrhagic Diseases. 19. Haemothorax. 20. Hand, Diseases and Deformities. 21. Headache. 22. Head Injury. 23. Heart Diseases—Methods of Investigation. 24. Heart Diseases—Congenital Diseases. 25. Heart Diseases—Innocent Murmurs. 26. Heart Diseases—Rheumatic Heart Disease in Children. 27. Heart Diseases—Aortic Valve Diseases. 28. Heart Diseases—Mitral Valve Diseases. 29. Heart Diseases—Myocardium Diseases. 30. Heart Diseases—Endocarditis, Bacterial. 31. Heart Diseases—Endocarditis, Chronic. 32. Heart Diseases—Right Side Diseases. 33. Heart Diseases—Heart Failure. 34. Heart Diseases—Pericardium. 35. Heat-Stroke and Heat-Exhaustion. 36. Heat Therapy. 37. Hemiatrophy and Hemihypertrophy. 38. Hemiplegia. 39. Hepato-Lenticular Degeneration. 40. Heredity and Constitution. 41. Hernia. 42. Herpes Zoster and Herpes Simplex. 43. Histoplasmosis. 44. Hodgkin's Disease. 45. Hydatid Disease. 46. Hydrocephalus. 47. Hydrotherapy. 48. Hyperchlorhydria. 49. Hyperidrosis. 50. Hypermetropia. Index to Volume Six.

Volume 6 of the second edition of B.E.M.P. continues to come up to the expectations which the previous volumes have given rise to.

Because of the alphabetical arrangement, there is much here that will appeal to the physician as well as to the surgeon, particularly as a very considerable portion of the volume is devoted to *Heart Diseases*. The contemporary leads for electrocardiography are described and illustrated fully.

Interesting is the very extensive revision which Sir Henry Tidy has made of the chapter on *Glandular Fever;* this name for the disease now receives much authority because it has been retained in the second edition. The classification of the clinical types has been altered to include juvenile, adolescent, anginose and long febrile types. The information about the Paul-Bunnell reaction is considerably amplified but there seems to be no reference to the important laboratory technique devised by Barrett in connexion with the serological diagnosis of the disease. The neurological manifestations receive special attention and the limited treatment available is set out very succinctly. Sir Henry deprecates the use of sulphonamides in this condition as the haemopoietic tissues are 'in a sensitive state'. He also emphasizes that antipyretics 'are contra-indicated in severe febrile cases as they aggravate the sweating and increase prostration'. He restricts the use of Penicillin to the anginose type.

The chapter on *Head Injuries* is extremely practical and South African colleagues will read with interest the authoritative account of *Hydatid Disease* prepared by Professor Dew, from Australia.

The printing and the binding are most attractive and the volume is a most useful contribution to this very impressive encyclopaedia.

THE KIDNEY: MEDICAL AND SURGICAL

The Kidney, Medical and Surgical Diseases. By Arthur C. Allen, M.D. (Pp. 583 + vii, with 1,115 illustrations. $15.00.) New York: Grune & Stratton. 1951.

Contents: 1. Introduction. 2. Embryology. 3. Anatomy. 4. Normal and Abnormal Physiology. 5. Uremia. 6. Malformations. 7. Diseases of the Glomeruli. 8. Diseases of the Tubules. 9. Interstitial Nephritis. 10. Specific Infections. 11. Pyelonephritis. 12. Renal Pelvic Calculi. 13. Renal Rickets. 14. Diseases of Vessels. 15. Tumors of the Kidney. 16. Histologic Artefacts. 17. Index.

Much ground has been covered since the urine-tasting days, but much about the kidneys still remains to be elucidated. With the development of the microscope and its application to the study of morbid anatomy, the hope was entertained that answers would be provided to the numerous problems encountered in the wards. It soon became apparent that there was not always a correlation between disturbances of function and morbid anatomical changes.

The author shows a clear insight into the discrepancies that arise from too facile pathological labeling, and attempts to narrow the gaps between ward, laboratory and autopsy room.

Very appropriately, a consideration of embryology, anatomy and of abnormal and normal physiology, constitutes the introductory chapters. The subjects are dealt with lucidly and their understanding assured by excellent illustrations. The author introduces his 'unitary theory' of nephron development. From histological studies he believes that rather than a union beween two separate portions (or failure of union in congenital cystic kidney), the metanephric diverticulum 'ramifies into the nephrogenic tissue, the active primordial, multi-potent cells of which are progressively added as epithelium corresponding to the cells of the advancing tubular buds until the entire length of the tubule is formed'. The principles of clearance studies are dealt with in a clear manner and their limitations pointed out.

The approach is a clinico-pathological one, but stress is laid on 'dynamic' pathology. This is not a compilation, but the result of critical observation and thought. Original opinion permeates accepted knowledge. Argument is offered why the focal lesion in subacute bacterial endocarditis is considered to be due to immuno-allergic responses rather than local bacterial action. In dealing with diseases of the glomeruli a new classification is introduced, based on morphological differences and implied closer correlation with clinical manifestations of oedema. This serves merely to emphasize the limitations of our knowledge and may increase confusion.

Almost every known condition with glomerular or tubular involvement receives individual consideration. The clinical picture and the diagnosis are dealt with clearly but concisely. Pathological description is supported by truly magnificent photomicrographs. The discussion of pathological physiology reflects the dynamic approach and an attempt at correlation. The book covers almost every possible entity of renal involvement, both common and rare. The text is illustrated with 1,115 superb photographs, a fifth of which deal with tumours of the kidney. Although these alone justify this publication,

it is more than an atlas; and the text, too, will repay careful reading. While some may not agree with all the views expressed by the author, one is impressed by the thought and experience from which these stem. This volume should find a place in every medical library.

TROPICAL MEDICINE

Clinical Tropical Medicine. By R. B. H. Gradwohl, M.D., L. B. Soto, M.D., and O. Felsenfeld, M.D. (Pp. 1647 + xxiii. With 473 illustrations and six colour plates. £9 11s. 3d.) St. Louis: The C. V. Mosby Company. 1951.

Contents: 1. Introduction to Tropical Medicine. 2. A General Consideration of the Protozoa. 3. Intestinal Protozoa of Man. 4. Amebic Dysentery, Hepatic Abscess, and other Intestinal Conditions caused by Protozoa. 5. Plasmodia of Man and Animals. 6. Malaria. 7. Malaria-Carrying Mosquitoes and their Control. 8. Chagas' Disease. 9. African Sleeping Sickness. 10. Leishmaniasis, Cutaneous, Mucocutaneous, and Visceral, with special reference to its occurrence in the Americas. 11 Pinta, Mal Del Pinto, or Carate. 12. Relapsing Fever. 13. Laboratory Diagnosis of Leptospiral Diseases. 14 Weil's Disease. 15. Fevers due to Rathite (including Sodoku). 16. Framboesia (Yaws). 17. The Viruses and Virus Diseases of Man—General Considerations. 18. Yellow Fever. 19. Dengue and Dengue-Like Fevers. 20. Psittacosis (Ornithosis, Psitacosis). 21. Encephalitis and Allied Diseases. 22. Lymphogranuloma Venereum. 23. Granuloma Inguinale. 24. Diseases due to Bacteria—Their Distribution and Occurrence in the Tropics. 25. Brucellosis. 26. Tularemia. 27 Plague. 28. Cholera (Asiatic Cholera). 29. Salmonellosis, Shigellosis. 30. Salmonellosis, Typhoid Fever, Shigellosis in Children. 31. Leprosy. 32. Human Bartonellosis or Carrion's Disease. 33. Tropical Phagedenic Ulcer (Vincent's Ulcer). 34. Respiratory Scleroma (Rhinoscleroma). 35. Rickettsioses or Rickettsial Diseases. 36. Rickettsialpox. 37. Helminthiasis. 38. Trichinosis. 39. Filariasis. 40. Onchocerciasis. 41. Dracontiasis or Guinea Worm Disease. 42. Cestodiasis or Diseases caused by Tapeworms. 43. Trematodiasis or Diseases caused by Flukes. 44. Fascioliasis Hepatica in Children. 45. Schistosomiasis Japonica. 46. Schistomiasis Haematobia and Schistosomiasis Intercalata. 47 Schistosomiasis Mansoni. 48. Tropical Mycosis—Introduction. 49. Superficial Tropical Mycoses. 50. Deep Tropical Mycoses. 51 South American Blastomycosis. 52. Histoplasmosis and Meliodosis. 53. Arthropods—Direct Agents in Tropical Diseases. 54. Myiasis. 55. Diseases caused by Ticks, Mites, Centipedes, and Lice. 56. Poisoning by Scorpions and Spiders (Scorpionism and Araneism). 57. Snake Venenation (Ophidism). 58. Poisoning by Fish and other Animals. 59. Diseases due to Nutritional Disturbances. 60. Sprue. 61. Beriberi. 62. Pellagra. 63. Cosmopolitan Diseases as seen in the Tropics. 64. Therapeutics of Tropical Diseases. 65. The Use of Antibiotics in Tropical Diseases. 66. Hygiene in the Tropics. 67. Organization of Preventive Medicine and Rural Hygiene Services in Sparsely Settled Tropical Countries. 68. Outline of Hematology as related to Tropical Diseases. 69. Histologic Technic in the Tropics. 70. Rapid Frozen Section Methods in Tropical Diseases. 71. Collection, Preservation and Examination of Stools in the Tropics. 72. Laboratory Methods in the Diagnosis of Helminthiases. 73. Reagents, Solutions, Stains, and Culture Media used in Parasitologic and Tropical Medical Procedures.

This book is a first edition of what will eventually be a most valuable reference book on tropical medicine for all workers in the tropics and subtropics. The three editors and their 57 assistants have undertaken a great task. Throughout the book the reader becomes aware of the fact that this team did not have scope enough and were feeling their respective ways along which to present their specialized knowledge, with the result that too many less relevant conditions have been included and too little of real tropical value has been given. We hope that this line of action will be more closely followed in the next edition.

The authors are mostly from the U.S.A., South America, India, East Indies, and Egypt. Thus informed opinion from tropical countries all over the world has been secured and made available to workers in tropical medicine everywhere. The diversity of authors has also led to a great diversity of approach and concept. Most of them are well-known teachers in tropical medicine. Some are younger men with ideas of their own, gained from daily experience in close contact with tropical diseases. Thus is introduced a welcome measure of new thought on some tropical conditions. The book therefore rightly pretends to be 'a record of specialistic knowledge'.

Special emphasis has been given to 'climatic, nutritional and transmissible diseases'. This makes the work valuable, for these are the very aspects of tropical medicine which are often neglected. In order to stress these, the authors have rightly not treated such conditions as small-pox, rabies, coccidioidosis and others. Similarly, such 'cosmopolitan diseases' as tuberculosis, pneumonia, mumps, poliomyelitis, hypertensive states, rheumatic fever, enterobiasis, and typhoid fever might, with advantage to the *treatment* of tropical diseases, have been deleted as well. Too wide a scope, if attempted in a work of this nature, must necessarily lead to lack of depth of treatment of the subject matter, thus detracting from the usefulness of the whole.

On the other hand, one appreciates the fact that such diseases as leprosy, yaws, tropical ulcers, the rickettsioses, filariasis, onchocerciasis, dracontiasis, schistosmiasis, tropical mycoses and snake venenation have been gone into fairly deeply. The chapters on tropical therapeutics are valuable, but could still be expanded considerably as this side of tropical medicine is what concerns the worker most often. Authoritative guidance about the use of the newer drugs against malaria, schistosomiasis and other conditions is what one seeks in a book of this nature. The same may be said of tropical hygiene and preventive medicine, with special reference to sparsely settled areas in the tropics. The evaluation of the sulphones in the treatment of leprosy might possibly be augmented. It would appear as if chaulmoogra and hydnocarpus oil is to a large extent still the treatment of choice in some areas; also that such measures as the local treatment of lepromata by means of carbon dioxide snow, and their cauterization by thermal, electrical and chemical means, still have their exponents in these days in which more human ways of treatment have become available. With respect to malaria, the question of early treatment *versus* 'therapeutic abstinence' is discussed and it is pointed out that allowing some 3-4 paroxysms to occur before treatment is instituted, is unwarranted and decidedly dangerous to the unfortunate patient. The drugs which still are the main-stay in the treatment of malaria at the present time are Mepacrine (Atebrin) and quinine. Paludrine is mentioned; but its curative value seems to be much inferior to that of the other drugs. This is in accordance with the reviewer's experience; Paludrine definitely is the best drug we have at present as a prophylactic against malarial infection provided it is used daily in adequate doses. It is disappointing to find that the valuable Ascoli treatment (by means of large and increasing doses of adrenaline—preferably with quinine—given intravenously) is not referred to. In the reviewer's hands this has proved very successful in the treatment of chronic and obstinate cases of malaria with enlarged spleens, as well as in cases which could not take the drug by mouth without vomiting. The gratifying success gained with Penicillin in the treatment of yaws is also referred to and it is to be hoped that the more painful bismuth-arsenical method of treatment will be supplanted by it. With respect to the treatment of schistosomiasis, the 1-day and 2-day treatment by means of sodium antimony tartrate introduced by Alves and Blair, is referred to as too dangerous for general use. In how far the treatment of bilharzia by means of Miracil-D (Nilodin), will establish itself, remains to be seen. It seems to be succesful in cases of haematobium but much less so in cases of mansoni infestation, and many patients have difficulty in retaining the large doses required to be taken by mouth.

No reference seems to have been made to the condition of malignant malnutrition. Perhaps this is only an inadvertent omission.

In scope and aim this book ranks next to Stitt's *Diagnostics and Treatment*, although much still requires to be added to the subject matter and much should preferably be deleted from the pages of this book, in order really to enable it to be what it aims to be, 'a record of specialistic knowledge'. In its present form, however, it nevertheless is a most helpful and practical reference book to all who have to cope daily with tropical conditions in Africa.

SURGERY OF CANCER

Nouvelles Techniques Operatoires Dans La Chirurgie Du Cancer. By Antonio Prudente and Henrique Melega. (Pp. 295, with 189 figures. 2,500 Fr.) Paris: Masson et Cie. 1951.

This book by two workers in South America deals with the radical surgical treatment of malignant disease. The work covers all regions and reflects considerable experience. It is very well illustrated and the French is sufficiently simple for anyone with a rudimentary knowledge of that language. This is a book that can well find a place in the library of every surgeon working in a large centre that deals with the problems of malignant disease, especially in its advanced forms.

STRUCTURE, FUNCTION AND EVOLUTION

Wonderfully Made. Some Modern Discoveries about the Structure and Functions of the Human Body. By A. Rendle Short, M.D., F.R.C.S. (Pp. 159 with 18 figures. 6s.) London: The Paternoster Press. 1951.

Contents: 1. The Wisdom of the Body. 2. The Human Foot. 3. Some Chemical Mechanisms of the Body. 4. The Digestive System. 5. Muscle and Muscular Exercise. 6. The Constancy of Body Temperature. 7. Repair. 8. Voice. 9. The Functioning of the Nervous System. 10. Functions of the Brain. 11. The Eye. 12. The Apparatus for Hearing and Balancing. 13. How the Embryo Develops. 14. The Problem of Man's Origin.

It is not the part of many outstanding scientists to take a decidedly Christian stand with respect to the pronouncements of science. In his many writings, intended for the layman, Professor Short discusses various issues in the light of modern knowledge and research and pays special attention to the implications of the theory of evolution. This one is another such little book.

The author this time takes the reader through the various regions of the human body, and discusses their anatomy and physiology very cursorily, but lucidly, using as few technical words and phrases as possible. He tries to demonstrate that we are truly 'wonderfully made' as to the structure and functions of our bodies; the outstanding wonders to him are the eye and the foot. With these facts to support him, Professor Short makes the point that blind chance could never have been able to achieve structure and function of such a degree of perfection—there must have been a Creator. Only an incorrigible agnostic will deny him this point.

If, however, one regards the infinitely small, but mighty, atom and thinks of the infinity of space and time, inexplorable as these are, one does find almost everywhere, e.g. in the vegetable and animal kingdom as well as in the histories of the past and the present races of mankind, in the stars, etc., many evidences of what, for lack of a clearer designation, one is apt to call *an evolutionary process which must have taken over at some time after the original creation.*

If one is not entitled to do this, because organic evolution still is a mere hypothesis, seemingly incapable of factual proof, then one is obliged to postulate repeated new acts of creation to account for the disappearance of old and the presence of new forms, that seem to have arisen during the passage of time. If there is no such thing as an 'evolutionary process' how should we account for the fact that all vertebrates have the same fundamental structure on which the science of Comparative Anatomy rests? The theories of Darwin, Wallace and dozens of others, do emphatically not constitute the 'theory of evolution' but are only so many attempts to try to explain its inner workings.

This book has at least this function, however, that it does tend to put a check upon too great a faith in human ability to explain what it fails to comprehend.

SEKSONDERRIG VIR OPGROEIENDE KINDERS

Mamma, Waar Kom Ek Vandaan? Deur Adriaan Smuts. (Pp. 37 met fotos. 5s.) Johannesburg: Afrikaanse Pers Boekhandel. 1950.

Hierdie boekie is 'n geslaagde poging om aan seksueel rypende kinders die grondsake van die voortplantingsproses in die lewe op eenvoudige wyse onder die aandag te bring. Te vele vind sulks langs onwenslike wee self uit.

Die skrywer laat uitkom dat beide die plante en diere ryke hulself voortplant deur gespesialiseerde selle afkomstig vanuit twee voorheen bestaande indiwidue—die ‚moeder' en die ‚vader'. Ons meen dat die woord ‚saad' nie sinoniem mag gebruik word met ‚eiertjie' nie. Laasgenoemde tog is uiteraard 'n *halwe* indiwidu, wat 'n ander helfte, van elders, moet bykry om 'n volledige indiwidu te word; terwyl eersgenoemde wel 'n volledige een van aard is en slegs op geskikte eksterne toestande wag om tot volle ontwikkeling te kom.

Die leser word vanaf die alledaags-waarneembare, bekende na die bedekte en nuusgierigheid-prikkelende, logies gelei.

Die uiteindelike eenheid van oorsprong van alle lewensvorms is 'n vername saak wat beklemtoon word. Ook is dit die geval met die feit dat soort, soort voortbring. Die *hoe* hiervan, meen ons egter, is bietjie te gevorderd vir kinders onder 16 jaar.

Hierdie boekie sal stellig 'n middel vir baie ouers wees om hulleself te red uit die monde van vraende kinders en wel op 'n waardige wyse.

CORRESPONDENCE

DISTRICT SURGEONS AND DRIVERS UNDER THE INFLUENCE OF ALCOHOL

To the Editor: As I am called upon to examine a good deal for alleged drunkenness, I read the letters from Drs. Lappin and Sive in your *Journal* a fortnight ago with interest, and I shall be obliged if you allow me to express a different point of view.

To regard the opinion of a district surgeon or other medical officer who examines a man alleged to be drunk as ridiculous —as does Dr. Lappin (13 October 1951)—is beyond me. Surely the examining medical officer is on account of his peculiar training, study and experience most fit to examine one so charged. He alone is capable of excluding such medical conditions as diabetes, hypertensive states, shock, etc., which simulate states of drunkenness often so closely. To arrive at a correct diagnosis in medicine one has often to perform 'odious' jobs, but I fail to see how the examination for alleged drunkenness is odious though I agree it is often thankless. Dr. Lappin says it is against a doctor's nature to condemn; but must the examining medical man condemn? Must he not rather give his honest and unbiassed opinion about what he found on examination?

Dr. Sive regards the examination of a person alleged to be drunk as 'a prostitution of a high calling'. On the contrary, one is often responsible, after a thorough examination, in saving a man from the loss of his job, his reputation or even a jail sentence when lay witnesses think, however fair-minded they are, that the accused is definitely drunk.

I experience a good deal of heartache in finding a person alleged to be drunk, definitely and unquestionably so.

As far as feeling humiliated because a magistrate differs from my opinion, unlike Dr. Sive, I rejoice that I am not the final judge whether the person I examined was indubitably drunk, but that it is left to the magistrate or judge to pass his judgment as he is better able to do so, having seen and heard many witnesses who, however fair-minded they may be, cannot leave out the emotional factor in their opinions, whereas the magistrate or judge makes allowance for it.

Kensington,
Johannesburg.
15 November 1951.

Daniel Jacobson.
Railway Medical Officer.

FEES IN PRIVATE PRACTICE

To the Editor: A report of a Special Committee of the Cape Western Branch recently expressed the view that if the maximum fee for general practitioners could be raised, it would help to raise their status.

While it has been found practicable to standardize the fees of Medical Aid Societies throughout the Union of South Africa, irrespective of the domicile of the beneficiary or the medical practitioner concerned, the standard fees of medical practitioners for consultations and visits still vary in different Branches.

If reasonable fees for surgical interventions in general practice are regarded as being 50% above Society rates, would it not be equally reasonable to apply this factor to the charges of general practitioners for consultations and visits, bearing in mind that the fee payable for a visit under the approved tariff is at present unreasonably low?

A high percentage of salaried employees are now eligible for membership of Aid Societies, and if it is the intention of the State to make provision for the lower income groups, who may at present be ineligible for such privileges, and hardship which might ensue through private medical fees being raised in proportion to the cost of living would be due to lack of forethought on the part of the public.

At all events, it has always been a doctor's privilege to reduce or waive fees according to the patient's financial circumstances, but despite social changes in the world, should

he not still be permitted to charge fees in keeping with inflation, and with his education and position in society?

The fees of specialists and dental surgeons are uniform throughout the country, as are the fees paid under the Workmen's Compensation Act. Thus, ample precedent exists for uniformity in the standard private fees of general practitioners.

Port Alfred. C. J. MacQuillan.
16 November 1951.

ULTRASONIC WAVES AND TREATMENT

To the Editor: In the Editorial of the *Journal* dated 22 September 1951, views are stated on the value and effect of treatment by ultrasonic waves. *Inter alia* reference is made to *Exerpta Medica*, Vol. 5, page 317, Section: Internal Medicine, 1951.* I have taken the trouble to look up this quotation but cannot find reference to the subject. As therapy by ultrasonic waves may be *terra nova* to most colleagues, I may be permitted to bring a few data, which together with reports from overseas medical journals and medical congresses, may throw some light on the subject. At the same time my letter will answer the Editorial.

In 1927 Wood and Loomis published a paper *Physical and Biological Effects of High Frequency Sound Waves of Great Intensity*. Since then about 3,000 publications have been made on the subject and not all of them in Western Europe. International Ultrasonic Congresses have been held in 1949 and 1950 at Erlangen and Rome attended by hundreds of internationally known physicists, biologists and physicians. Well-known firms in the U.S.A. and in Germany have developed, during the last 10 years, reliable ultrasonic apparatus which is to-day in the hands of thousands of physicians. Ultrasonic treatment has been put on the list of German Government Panel Tariff of Fees alongside with Diathermy and X-ray therapy. We all know how careful such organizations are to approve of new methods of therapy.

Ultrasonic therapy is admittedly a new form of energy. The waves are not electromagnetic, but simply mechanical and produce rhythmical concentration and relaxation of the tissues which they penetrate. In chemistry ultrasonic waves have been used for many years to produce colloids. In the living organism ultrasonics produce heat by their rhythmical impact, they easily seem to penetrate tissues of an equal density, but seem to concentrate heat, when they meet, e.g. bone or nerves or tendons. Ultra-short waves seem to come nearest to this effect, whereas infra-red and heat applications will only affect the outer tissues. Research has not been able to decide yet, whether the chemical or the mechanical effects of ultrasonic waves have brought relief of pain; probably both. Good results have been found in the treatment of:

1. Neuralgias (sciatica, lumbago, plexus and intercostal neuralgia).
2. Chronic arthritis, arthritis deformans, Bechterew. (Naturally bony changes or exostosis cannot be removed, but patients mostly feel great relief after few treatments.)
3. Myalgias.
4. Bronchial asthma.
5. Ulcera cruris, X-ray ulcera, trophic ulcera.
6. Furunculi, carbunculi, mastitis, lymphadenitis.
7. Prostatitis.
8. Morbus Buerger and other circulatory derangements.

The dangers, which have been pointed out in the Editorial, are not really greater than those caused by X-ray and diathermy, when the apparatus is being used under strictest medical supervision. Besides, ultrasonic waves have the most effective danger signal: pain. If ultrasonic treatment is kept below the pain limit, it should (according to the findings of research workers) not produce any ill effect in the tissues. In no circumstances should the abdominal region be treated in cases of pregnancy. Patients suffering from heart diseases do not seem to feel comfortable, when stronger ultrasonic energies are being used in the region of the heart. Patients who have marked myopia are also not suitable for treatment of the neuralgias of the head. In the U.S.A. tests have been made, where bones and tissues of young dogs have been subjected to long treatment by strong energies of ultrasound waves. The ill effects caused by such energies can most certainly not be produced by the carefully developed apparatus of well-known and reliable firms, which use only a few watts of current.

The enthusiastic reports on ultrasonic therapy and its results from all over the world seem to be proof enough that medical science has been given a new weapon to fight disease and pain.

373 Main Street, B. M. Kranz.
Paarl.
21 October 1951.

* [Owing to an unfortunate printer's error, the page reference was given as 317 instead of 17.

Our correspondent, however, appears to have missed the point of the Editorial, which stressed the dangers of allowing ultrasonic apparatus to be used by inexpert lay persons. The Editorial also emphasized that at present there was no sound evidence of any *specific* therapeutic virtue inherent in these waves. Claims of clinical benefit were not denied but attention was drawn to the need to regard these claims as still being in the field of experiment and research rather than daily practice.—*Editor.*]

To the Editor: In view of the eminently reasonable and cautious views on the use of ultrasonic waves in treatment expressed in your Editorial of 22 September, your readers may be interested in the following extract from the *British Journal of Physical Medicine* of June 1951.

'In an annotation in the *Lancet* of 17 February 1951, it is pointed out that the British Ministry of Health has limited the provision of ultrasonic emittors to specified hospitals, in which facilities for critical physical and clinical trials can be carried out. Attention is drawn to the very large claims, covering an extremely wide field of indications, which have been made for the value of the method, particularly in Germany and Austria.

One medical patient of our acquaintance, who was subjected to it, remarked afterwards that its inventor ought to be hanged with it; but neither individual failures nor individual successes are any sounder proof of the value of a method than is the proverbial single swallow as a harbinger of summer. The Athenians were said to be noted for their enthusiastic reception of any novelty. Perhaps this part of the legacy of Greece has descended to too many heirs.'

The Long and Short of It.
16 November 1951.

THE AETIOLOGY OF LUNG CANCER

To the Editor: The increasing incidence of carcinoma of the lung is again receiving ratiocinative discussion in medical circles. With your kind permission I would like to briefly mention some original work on an hitherto unconsidered factor in the causation of this dread disease.

Historically, a virtually unconsidered clinical entity in the 19th century has almost the appearance of a Moloch in the mortality statistics of the second half of the 20th century. And the cause of it all lies under our very noses, literally under our very noses—for the culprit, Sir, is face powder.

A graphic comparison of the incidence of lung cancer and the sales of face powder exhibits virtually parallel curves with an almost vertical rise in the last decade.

An apparent anomaly is the fact that the sex making most use of this agent is less susceptible to its ravages. A visit to any night club will explain this paradox. Women make infinitely more use of cosmetics than the male section of the population and, therefore, superficially one would expect this disease to be more common in the female sex. But careful thought shows that the area of application to which a woman exposes herself by inhalation is only approximately one square inch below her ala nasae, and even that area is not continuously so covered by the carcinogenetic agent. When the male is periodically exposed, particularly at certain times when the intervening distance between the female integument and the male ala nasae can only be measured in fractions of an inch, a coated area is involved consisting of the entire face and neck and, latterly, with the advent of more daring fashions, it even includes the back and shoulders of the female. Moreover, the female, whilst in male company, renews the application of this toxic preparation as rapidly as it is inhaled, or removed

by ingestion. Is it any wonder then that the incidence is so much higher in the male than in the female? A comparison of the time-exposure-area factors of both sexes is proportionately consistent with the higher sex incidence. Confirmatory evidence is available in the fact that no pulmonary carcinoma has been reported in any male with a hirsute appendage of the upper lip with a horizontal growth of more than 2.25 cm. and a density of more than 218 hairs per square millimetre. Mouth breathers were excluded in this survey.

I would like to mention two more statistical points of interest. A colleague of mine who has been practising among the Bamangulu for the last 53 years confirms that to date he has not yet seen a single case of lung carcinoma in the tribe. Owing to involved tribal taboo, face powder is not used by them. Also, during the last war, an analysis of the mortality rate of the front-line troops in action on both sides showed not a single death due to pulmonary carcinoma, despite the millions involved. Obviously the absence of women in the firing line entailed a concomitant absence of this carcinogenetic agent.

From my researches I can safely predict that in the coming decade female incidence of this disease will increase with the more frequent use by the male of after-shave talc, etc.

These few points extracted from a monograph on which I am now engaged will perhaps assist the profession to better evaluate aetiological factors in the production of pulmonary carcinoma.

REFERENCE

Principles of Medical Statistics, Bradford Hill, 5th ed., 1950, pp. 1–242.

P.S.—On a recent visit to Johannesburg some respected colleagues pointed out similar facts regarding possible results of tobacco smoking. Whilst I consider my own findings conclusive, I am a smoker and, cogitating on their remarks, my nerves have become upset. I am now smoking twice as much. Could you perhaps recommend a good psychiatrist?

E. Baskind.

The Rooiberg Minerals Development Co. Ltd.,
P.O. Rooiberg,
Transvaal.

MEDICAL AID SOCIETIES

To the Editor: The Member Societies of this Council have now had the opportunity of studying the new Tariff of Fees which will operate from 1 January 1952, in regard to its effect on their own financial position and the additional cost which the individual member will have to bear. The Tariff was presented by the Medical Association to representatives of Medical Aid Societies on 2 November 1951, at a joint meeting which had been postponed from June when earlier discussions on the new schedule had taken place.

It was understood by this Council that the Meeting had been called to negotiate a new Tariff but we were informed that as Federal Council had accepted the Tariff, no variation in the fees could be discussed. The Societies were, therefore, faced with an accomplished fact, whereas previous Tariffs had been the result of mutual agreement.

After close and prolonged deliberations the following Societies have decided to withdraw as approved bodies:—

1. Atlantic Refining Company Staff Medical Aid Society.
2. Caltex Medical Aid Society (South Africa).
3. Cape Times Medical Aid Society.
4. Siektefonds van die Nasionale Pers, Beperk (Kantoorpersoneel).
5. Norwich Union Life Insurance Society Staff Medical and Surgical Benefit Scheme.
6. S.A. Teachers Association Medical Aid Society.
7. S.A.K.A.V. Sick Benefit Fund.
8. S.A. Mutual Life Assurance Society Staff Medical Aid Fund.
9. The Southern Medical Aid Society.
10. United Banks Medical Aid Society.
11. Vacuum Medical Aid Society (South Africa).

From 1 January 1952 these Societies will cease to have direct relationship with medical practitioners and doctors' accounts will not be paid direct. Whatever benefits are due to the member, based on the individual Society's own Tariff, will be paid to him direct.

These Societies represent 40,000 individuals throughout the Union and disbursed approximately £175,000 during the past year to doctors. The amount paid out is the full Tariff fee without any deduction for the percentage which has to be borne by the Members. The recoupment from the member of his share of the fees has been undertaken by the Society.

This Council submitted a Tariff to the Central Contract Practice Committee for its consideration but it was rejected. It is the opinion of the Council that its Tariff was fair for both doctor and patient and within the financial resources of Societies. It was not prepared without consulting several eminent practitioners and it provided for differential fees of 11s. 6d. and 10s. 6d. respectively for General Practitioners' visits and consultations.

In conclusion the Societies concerned would like to express their sincere appreciation to all medical practitioners who have so willingly co-operated with them over many years.

A. C. Sargeant,
Secretary.

The Southern Council of Medical Aid Societies,
78 Hout Street,
Cape Town.
23 November 1951.

[*The Medical Secretary writes*: When the Medical Association of South Africa agreed to a reduction of 10 per cent in the Tariff of Fees for Approved Medical Aid Societies, dating from 1 July 1950 and lasting for one year only, it did so at the request of the Medical Aid Society representatives and on the clear understanding that the Tariff would be subject to review at the end of that period. When the full fee payable to general practitioners was restored at the beginning of this year, it was done so at the instance of the Medical Aid Societies and not through any request on the part of the Federal Council of the Association. In the early months of this year the various Branches and Groups were asked to submit suggestions for the Tariff of Fees and it was pointed out, as is usual, that the fees so submitted should be preferential in character as they are applicable to the so-called lower middle income groups who form the majority of the membership of Medical Aid Societies.

A copy of the Tariff based on these suggestions was circulated to all approved Medical Aid Societies in May of this year and representatives of the Societies were invited to attend a joint meeting with members of the Central Committee for Contract Practice on 6 June in Johannesburg. At that meeting representatives of the various Specialist Groups were asked to be present to state the case for their Groups and to hear the counter-arguments put up by the Societies' representatives. At the end of the meeting agreement had been reached concerning a large number of points raised by the Societies, notably in the Preamble to the Tariff, but also as regards certain specific fees. The representatives stated that they would have to return to their Societies to discuss the matter further and the Association's members agreed to take up the points of difference with the Groups concerned. This was done by the Association and a further meeting was called for 2 November. Before this meeting, however, the replies of the Groups had been considered by the Central Committee for Contract Practice and the Federal Council, and the Tariff remained almost identical with that discussed with the Societies' representatives in June. At the November meeting further alterations were made in the Preamble to the Tariff at the request of a representative of a well-known Medical Aid Society, but the Central Committee for Contract Practice had no authority to alter the fees as these had already been referred back and had been finally accepted by the Council.

It would seem that a number of the Medical Aid Societies formerly approved by the Association have now decided to contract out of the Tariff, and members are advised that they are under no obligation to treat members of these Societies (i.e. all Societies not listed in the new Tariff Book which is shortly to be published) at preferential rates, and it would be in order for such persons to be charged ordinary customary fees.

It is possible that certain Societies may attempt to negotiate with medical practitioners to supply services on some other tariff basis, but the Association would regard such an agreement entered into by a medical practitioner as unethical.]

The Medical Association of South Africa
Die Mediese Vereniging van Suid-Afrika

AGENCY DEPARTMENT : AGENTSKAP AFDELING

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(I O27) Examination couch, £7 10s.

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KAAPSTAD : CAPE TOWN

Posbus 643, Telefoon 2-6177 : P.O. Box 643, Telephone 2-6177

PRAKTYKE TE KOOP : PRACTICES FOR SALE

(877) Cape Midlands. Exceptionally well-established solus prescribing practice in progressive inland hospital town. Good deal of surgery done. Average annual receipts approx. £6,200. One appointment. Premium required £3,000. House for sale at £4,000. Terms could be arranged. Good schools. Willing to give thorough introduction.
(848) Cape Town, Southern Suburbs. Solus practice. Average annual receipts £2,088. Premium and terms open to discussion. Excellent opportunity.
(860) Noordelike Kaapprovinsie. Vooruitstrewende aangename hospitaaldorp met uitstekende skole. Venootskapspraktyk met kontant inkomste van £6,459 in afgelope jaar. Twee aanstellings. Uitstekende geleentheid vir snykunde.

VENNOOTSKAP VERLANG : PARTNERSHIP REQUIRED

(811) Partnership share in Cape or Natal in predominantly English-speaking practice with min. net income £2,500 p.a.

ASSISTENTE/PLAASVERVANGERS VERLANG
ASSISTANTS/LOCUMS REQUIRED

(887) Cape Town suburb. Assistantship for 1 year from 1 February or as soon thereafter as possible. Salary offered £2 2s. a day plus all found and possibly extra emoluments. It is not necessary for the assistant to possess his own car.
(809) Gentile assistant for Transkei general practice with D.S. appointment. Single man preferred. Excellent opportunity to gain sound experience. Salary to be arranged.
(406) Assistant who wishes to gain experience in anaesthesia. Woman preferred. Salary to be arranged.
(826) Cape Town general practice. 1 January for 3 weeks. £3 3s. p.d. plus all found. Experience of anaesthetics required.
(869) Medical officer for Tristan da Cunha. Free quarters with hard furnishings also passage to and from Cape Town. Salary £1,200 per annum, 3 months' notice. Married man preferred.
(847) Noord-Kaapland. Vanaf ongeveer 20 Desember vir 6 tot 8 weke teen 2 tot 3 ghinies p.d. plus kartoelae en reis en ander kostes.

MEDICAL EQUIPMENT FOR SALE

(772) Strand, C.P. Couch, instrument and dressing tables, cupboards and waiting-room furniture, at approx. £100. Instruments at £100.
(758) Electrocardiograph. Sanborne Cardiette. Weight 24 lb. Perfect working condition. Used by Cape Town specialist physician. £160 or nearest offer.
(674) *British Encyclopaedia of Medical Practice.* Any reasonable offer.
(878) White wooden cabinet. Five feet high. Top half glass doors and shelves. £23 10s.

ASSISTENTE/PLAASVERVANGERS BESKIKBAAR
ASSISTANTS/LOCUMS AVAILABLE

(784) Lady doctor qualified Univ. C.T. 1944, additional experience Paediatrics, available as locum, assistant or Junior Partner in Port Elizabeth or environs.

CONSULTING ROOMS REQUIRED

(907) Cape Town. Two rooms and share waiting room and services of nurse/receptionist. Urgent.

Frontier Hospital

QUEENSTOWN

Medical practitioners who have been in practice in Queenstown during the past 12 months, are invited to apply for appointment to the Honorary Medical Staff of the Frontier Hospital.

Applications to be sent to the Medical Superintendent on or before 22 December 1951.

Frontier Hospital (3771)
29 November 1951

For Sale

One Gomco anaesthetic and suction apparatus (Gomco pump), latest model, electrically operated, ideal for minor surgery and throat work. £80 or nearest offer.

One Bircher diathermy set, cables, pads, fitted for electrosurgery and all newest developments, latest model, AC/DC, £100 or nearest offer.

One Reichert research microscope, single and double eyepieces, movable stage, oil immersion, etc., latest model imported, £100 or nearest offer.

Write to 'A. J. L.', P.O. Box 643, Cape Town.

City of Kimberley

LOCATIONS MEDICAL OFFICER

Applications are hereby invited from qualified registered medical practitioners under 45 years of age, for the post of Medical Officer (Clinical) in the Council's Native Locations on the grade £800 - 50—£1,000 per annum, plus temporary cost-of-living allowance. The commencing salary will be determined by qualifications and experience.

The successful applicant will be in charge of the Locations Medical and Nursing Service, under the jurisdiction of the Medical Officer of Health and will carry out such duties as the Medical Officer of Health may determine. The appointment will be subject to the Municipal conditions of service.

Applications, stating age, qualifications, experience and the earliest date duty can be assumed and accompanied by copies of not more than three recent testimonials, must reach the undersigned not later than 22 December 1951.

Town Office
Kimberley
28 November 1951
No. 206/1951

R. Hartley Marriott
Town Clerk
(1414)

Municipality of Somerset East

NOTICE NO. 57/1951

VACANCY FOR PART-TIME MEDICAL OFFICER OF HEALTH

Applications are invited from suitably qualified candidates for the abovementioned post at a salary of £180 per annum. No cost-of-living or other allowance is paid.

The successful applicant will be required to enter into an agreement with the Council, copy of which may be seen at the office of the undersigned.

Applications, giving full particulars of qualifications, experience, marital state, age and nationality, should be submitted to the undersigned not later than 5 January 1952.

Somerset East
15 December 1951

P. J. Swanepoel
Town Clerk
57/1951

New Germany Town Board

PART-TIME MEDICAL OFFICER

Applications are hereby invited for the post of part-time Medical Officer of Health for this Township, on terms and conditions to be arranged.

For further particulars please enquire from the undersigned, with whom applications must be lodged at this office not later than 11 a.m. on Saturday, 22 December 1951.

Town Board Office (Phone 7-6418)
P.O. New Germany
Natal

T. G. Schmidt
Town Clerk
7/4

For Sale

Well-established practice in Eastern Free State with modern hospital and maternity home facilities. Gross income for previous 12 months £4,700. Price £2,000, including complete stock of instruments, medicine and furniture. Apply to 'X. Y. Z.', 1 Browne Street, Bloemfontein.

Te koop

Goed gevestigde Oostelike Vry-Staatse praktyk met moderne hospitaal en Kraaminrigting fasiliteite. Gros inkomste afgelope 12 maande £4,700. Prys: £2,000, insluitende volle voorraad instrumente, medisyne en meubels. Skryf aan 'X. Y. Z.', Brownestraat 1, Bloemfontein.

Trained Sister

Efficient and hardworking, with wide experience in medical and clerical work, requires position as receptionist with one or more medical practitioners or as Staff Sister for an industrial concern. Write to 'A. J. N.', P.O. Box 643, Cape Town.

Provincial Administration of the Cape of Good Hope

HOSPITALS DEPARTMENT

ELECTION OF MEDICAL COMMITTEES

The attention of registered medical practitioners is drawn to the Regulations relating to the election, powers and functions of Medical Committees of Provincial Hospitals, framed in terms of sections 33 and 52 of Ordinance No. 18 of 1946, as amended.

The election of Medical Committees for the following institutions will take place on a date to be fixed in March 1952:—

Groote Schuur Hospital.
Somerset Hospital.
Woodstock Hospital.
Rondebosch and Mowbray Hospital.
Victoria Hospital, Wynberg.
False Bay Hospital, Simon's Town.
Peninsula Maternity Hospital. } Joint
Mowbray Maternity Hospital. } Medical
Maternity Section, Somerset Hospital. } Committee.
Lady Michaelis Orthopaedic Home, Plumstead.
Princess Alice Home of Recovery, Retreat.
Cape Town Free Dispensary.
Conradie Home, Pinelands.

Every registered medical practitioner who has been in active practice for at least two years in the areas normally served by the above institutions, and who has attended two or more patients in the hospitals within the twelve months preceding the election, is eligible to vote.

Prescribed registration forms are obtainable from the Medical Superintendents of the institutions mentioned, and medical practitioners desirous of being registered should forward such forms, countersigned by the Medical Superintendent, to the *Chairman of the Hospital Board concerned*, P.O. Box 1487, Cape Town, not later than noon, on 31 December 1951.

The following are the Hospital Boards dealing with the abovementioned institutions:—

Teaching Hospitals Board (Cape Town): Groote Schuur Hospital; Peninsula Maternity Hospital; Mowbray Maternity Hospital.

Somerset Hospital Board: Somerset Hospital.

Southern Suburbs Hospitals Board: Woodstock Hospital; Rondebosch and Mowbray Hospital; Victoria Hospital, Wynberg; Lady Michaelis Orthopaedic Home; Princess Alice Home of Recovery; Cape Town Free Dispensary.

False Bay Hospital Board: False Bay Hospital.

Northern Areas Hospital Board: Conradie Home.

L. Welham
Branch Representative
(12195)

58 Loop Street
Cape Town

Medical Officer

Applications are invited from medical practitioners in Alberton for the position of panel doctor to the undermentioned Society. Conditions of appointment will be in terms of the requirements of the Medical Association of South Africa. Please reply with full particulars to: The Secretary, Alpha Harris Benefit Society, P.O. Box 24, Knights.

Medical Officer

Applications are invited from medical practitioners for the position of panel doctor to the undermentioned Society in the Bedfordview-Edenvale area. Conditions of appointment will be in terms of the requirements of the Medical Association of South Africa. Please reply with full particulars to: The Secretary, Alpha Harris Benefit Society, P.O. Box 24, Knights.

Provincial Administration of the Cape of Good Hope

UNIVERSITY OF CAPE TOWN: JOINT MEDICAL STAFF FOR GROOTE SCHUUR AND OTHER TEACHING HOSPITALS: VACANCIES

Applications are invited from registered medical practitioners for appointment to the undermentioned posts:—

Groote Schuur Hospital: Department of Physical Medicine.

Post	*Salary*
1 post of medical practitioner, Grade F	£1,800 per annum
1 post of medical practitioner, Grade C	£1,000 × 50—£1,200 per annum

The post of medical practitioner, Grade F, will be filled in a part-time capacity on a sessional basis. A total number of 11 sessions are available and the remuneration for each session (of 4 hours per week) shall be at the rate of £164 per annum.

The post of medical practitioner, Grade C, will be filled in a whole-time capacity on contract.

In addition to the scale of salary indicated, a cost-of-living allowance at rates prescribed from time to time by the Administrator is payable to persons employed in a whole-time capacity. (Present rate is £256 per annum for married and £80 per annum for single persons.)

The following are the minimum requirements for appointment to the abovementioned posts:—

Post medical practitioner, Grade C: Not less than 5 years' experience after graduation or four years' experience after registration, of which not less than 3 years shall have been spent in training as a specialist in physical medicine.

Post medical practitioner, Grade F: Not less than 3 years' experience after registration of a specialist in physical medicine.

The following conditions of service will apply to appointments to the Joint Medical Staff:—

(a) All appointments will be in terms of and subject to the provisions of Ordinance No. 19 of 1941, as amended, and the regulations framed thereunder.

(b) The Joint Medical Staff will be required to serve jointly the Provincial Administration of the Cape of Good Hope and the University of Cape Town.

(c) A session shall be 4 hours per week, not necessarily continuous of clinical and/or teaching work.

(d) Attendances at staff conferences and Medical Committee meetings will constitute part of the duties but will not be regarded as sessional attendances for which payments are made to part-time employees, as such attendances will, like attendances for emergencies, be regarded as covered by the ordinary sessional payments.

(e) Candidates for the Grade F post must state the maximum number of sessions which they would, on appointment be prepared to give, indicating preference for days and times.

(f) Contracts for the Grade C post will be up to a maximum period of 4 years' appointment to be subject to confirmation at the end of the first 12 months and further subject to termination at any time on 90 days' notice on either side.

The successful candidates who are not already in the Hospital Board Service, will be required to submit satisfactory birth and health certificates.

Application must be made on the prescribed form (Staff 23) which is obtainable from the Director of Hospital Services, P.O. Box 2060, Provincial Building, Wale Street, Cape Town, or from the Branch Representative of the Hospitals Department at Cape Town (P.O. Box 1487), Port Elizabeth (P.O. Box 80), East London (P.O. Box 13), Kimberley (P.O. Box 618) and Umtata (P.O. Box 202) or from the Medical Superintendent of any Provincial Hospital or Secretary of any School Board in the Cape Province.

The completed application forms must be addressed to the Director of Hospital Services, P.O. Box 2060, Cape Town, and must reach him not later than 18 January 1952. (Y 249872)

Provinsiale Administrasie van die Kaap die Goeie Hoop

UNIVERSITEIT VAN KAAPSTAD: GESAMENTLIKE MEDIESE PERSONEEL VIR GROOTE SCHUUR-HOSPITAAL EN ANDER OPLEIDINGSHOSPITALE: VAKATURES

Aansoeke word ingewag van geregistreerde geneeshere vir aanstelling in die ondergenoemde poste:—

Groote Schuur-hospitaal: Departement van Fisiese Geneeskunde.

Pos	*Salaris*
1 pos van geneesheer, Graad F	£1,800 per jaar
1 pos van geneesheer, Graad C	£1,000 × 50—£1,200 per jaar

Die pos van geneesheer, Graad F, word in 'n deeltydse hoedanigheid op 'n sessie-basis aangevul. Daar bestaan 'n totale getal van 11 sessies en die besoldiging vir elke sessie (van 4 uur per week) bedra £164 per jaar.

Die pos van geneesheer, Graad C, word in 'n voltydse hoedanigheid op kontrak aangevul.

Benewens die salarisskaal soos aangedui, is 'n lewenskostetoelae teen tariewe wat van tyd tot tyd deur die Administrateur vasgestel word, betaalbaar aan persone wat voltyds in diens is. (Die huidige tarief is £256 per jaar vir getroudes en £80 per jaar vir ongetroudes.)

Die volgende is die minimum vereistes vir aanstelling in die bogenoemde poste:—

Pos geneesheer, graad C: Minstens 5 jaar ondervinding na ontvangs van graad of 4 jaar ondervinding na registrasie, waarvan minstens 3 jaar aan opleiding as 'n spesialis in fisiese geneeskunde bestee is.

Pos geneesheer, Graad F: Minstens 3 jaar ondervinding na registrasie as 'n spesialis in Fisiese Geneeskunde.

Die volgende diensvoorwaardes is van toepassing op aanstellings in die gesamentlike mediese personeel:—

(a) Alle aanstellings is ingevolge en onderworpe aan die bepalings van Ordonnansie nr. 19 van 1941, soos gewysig, en die regulasies daarkragtens opgestel.

(b) Van die gesamentlike mediese personeel word vereis om die Provinsiale Administrasie van die Kaap die Goeie Hoop en die Universiteit van Kaapstad gesamentlik te dien.

(c) 'n Sessie is 4 uur per week in verband met kliniese en/of opleidingswerk maar is nie noodwendig onafgebroke nie.

(d) Bywoning van personeelkongresse en mediese komiteevergaderings maak deel uit van die werk maar word nie beskou as sessiebywonings waarvoor deeltydse werknemers betaling ontvang nie, aangesien daar beskou word dat sodanige bywonings gedek word deur die gewone betaling vir sessies op dieselfde wyse as bywonings vir noodgevalle.

(e) Kandidate vir die pos Graad F moet die getal sessies vermeld wat hulle by aanstelling gewillig is om diens te doen asook die dae en tye.

(f) Kontrakte vir die pos Graad C is vir aanstelling vir 'n maksimum tydperk van 4 jaar, en die aanstelling is onderworpe aan bekragtiging aan die einde van die eerste 12 maande en verder onderworpe aan opsegging te eniger tyd na kennis van 90 dae van die een of die ander kant.

Die geslaagde kandidate wat nie reeds in die hospitaalraadsdiens is nie, moet bevredigende geboorte- en gesondheidsertifikate indien.

Aansoek moet gedoen word op die voorgeskrewe vorm (Staf 23) wat verkrygbaar is by die Direkteur van Hospitaaldienste, Posbus 2060, Provinsiale Gebou, Waalstraat, Kaapstad, of by die Takverteenwoordiger van die Hospitaaldepartement te Kaapstad (Posbus 1487), Port Elizabeth (Posbus 80), Oos-Londen (Posbus 13), Kimberley (Posbus 618), en Umtata (Posbus 202), of by die Mediese Superintendent van enige provinsiale hospitaal of by die sekretaris van enige skoolraad in die Kaapprovinsie.

Die ingevulde aansoekvorms moet aan die Direkteur van Hospitaaldienste, Posbus 2060, Kaapstad, gepos word en moet hom nie later as 18 Januarie 1952 bereik nie. (Y 249872)

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